The American Journal of Surgery

NEW SERIES, VOL. XXI AUGUST, 1933 No. 2

THERAPEUTIC EFFECT OF COMPLETE THYROIDECTOMY ON CONGESTIVE HEART FAILURE & ANGINA PECTORIS

IN PATIENTS WITH NO CLINICAL OR PATHOLOGICAL EVIDENCE OF THYROID TOXICITY*

II. OPERATIVE TECHNIQUE†

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THE purpose of this communication is to outline the technical steps involved in the total ablation of the normal thyroid gland on a group of 11 patients with congestive failure or angina pectoris. The first case of this series has been described in a previous communication.[1] The clinical results in the other 10 patients will be reported in a forthcoming communication.[2]

Beginning his researches on the velocity of blood flow early in 1925 at the Thorndike Memorial Laboratory, Blumgart and his co-workers[3] demonstrated that there was a close correlation between the demands of the tissue metabolism and the basal speed of blood flow; the higher the metabolic rate, the greater the demands on the heart. For example, in patients in whom the basal metabolic rate was elevated to approximately plus 30 per cent, the speed of blood flow was found to be twice the normal. In myxedema, on the other hand, with the metabolism correspondingly depressed, the velocity of the blood flow was reduced to approximately one-half that of normal. In patients with circulatory failure, the velocity of blood flow was considerably slower than in noncardiac patients with a similar basal metabolic rate.[4]

This important relationship between the demands of tissue metabolism and the supply of blood by the normal heart formed a valid theoretical basis for believing that in patients with chronic heart disease and a normal metabolic rate the production of artificial myxedema might result in striking clinical improvement. The hearts of such persons might be unable to supply sufficient blood for the ordinary demands of a normal metabolic rate, but might, nevertheless, be able to supply enough blood for the lessened needs of a reduced metabolic rate. Clinical improvement of the circulatory function of thyrocardiac patients after subtotal thyroidectomy, demonstrated by Kocher,[5] Hirschfelder,[6] Hamilton,[7] Levine and Sturgis,[8] White,[9] Lahey,[10] Rosenblum and Levine[11] and the well-recognized value of a reduction in metabolic rate by rest and

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† Part I of these studies has already been published[1] and Part III will appear soon.[2]

sedatives[12] were in harmony with these considerations.

Before the present series of operations of total ablation of the thyroid was undertaken, 2 patients suffering from congestive heart failure were operated upon in October and November, 1932. Approximately nine-tenths of the gland was removed. Both patients made excellent operative recoveries and showed striking clinical improvement, which paralleled the fall in the metabolic rate. Despite this very extensive but incomplete removal of the gland, the metabolic rate began to rise during the fifth week after operation and the clinical improvement, as might be expected, became less pronounced. A study of the results in these 2 patients suggested to Blumgart the desirability of performing a total ablation of the thyroid gland in order to establish the necessary persistent lowering in the metabolic rate.[1]

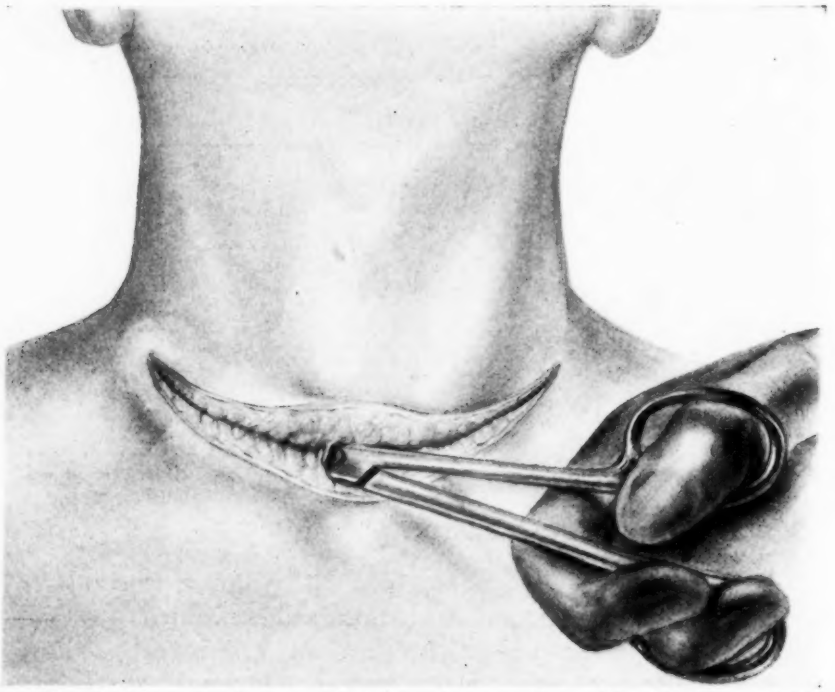

FIG. 1. Low collar incision. Beginning reflection of upper flap with aid of curved Mayo scissors.

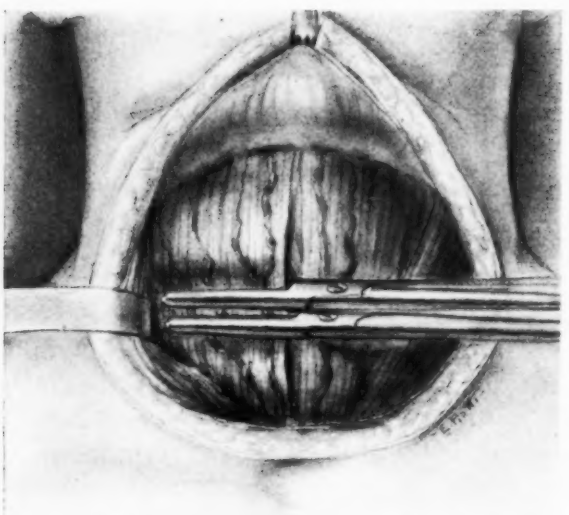

FIG. 2. Pre-thyroid muscles exposed and clamped between Ochsners on the right side, preparatory to transection.

After a study of the special surgical considerations involved, I performed the first total ablation of the thyroid gland on patient G. F. on December 15, 1932.[1] During the six months that have elapsed since this operation, the metabolic rate has remained persistently low, averaging minus 28 per cent. The patient is now restored to occupational usefulness after three years of complete cardiac invalidism. As far as can be ascertained, this is the

first time that a total ablation of the normal thyroid gland for chronic heart disease of non-thyrogenous origin had ever been performed.

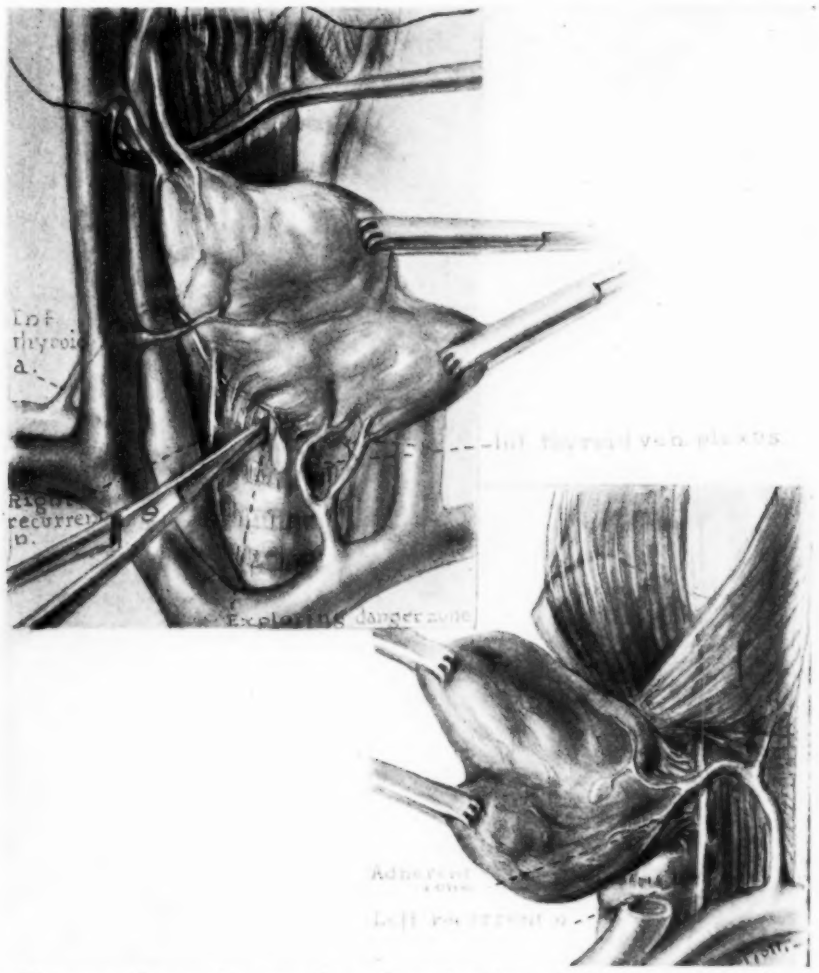

Fig. 3. (*Upper.*) Ligature of superior vessels and exploration of danger zone with fine blunt scissors showing parathyroid gland and recurrent nerve coursing anterior to lower terminal branch of inferior thyroid artery (right side). (*Lower.*) Adherent zone, indicating fixation of gland to trachea and of times to recurrent nerve. Latter is shown passing posterior to artery (left side).

Radical, or so-called total, thyroidectomy of the hyperplastic gland in thyrotoxicosis, as reported by Gilman and Kay,[13] is not an infrequent procedure today. These authors removed as much of the hyperplastic gland as was surgically feasible, "shaving the posterior portion of each lateral lobe as close as possible to the capsule or by stripping the capsule from the posterior surface of the lobe." For our purposes, even this would not be adequate as indicated by our first 2 cases, since it would not insure the desired permanent lowering of the metabolic rate and the associated persistent clinical improvement.

It is fortunate that those suffering from chronic heart disease, even with congestive failure, tolerate such surgery reasonably well. These patients are far from ideal surgical risks, and, it may be said, experience in thyroid surgery together with an accurate knowledge of the regional anatomy is of primary importance to the operator. Since the patients are not thyrotoxic, they do not present the added element of toxicity. For this reason, a one-stage operation has been satisfactorily performed under gas-oxygen analgesia with

preliminary morphia and sodium amytal medication.

Surgery of the thyroid gland has been

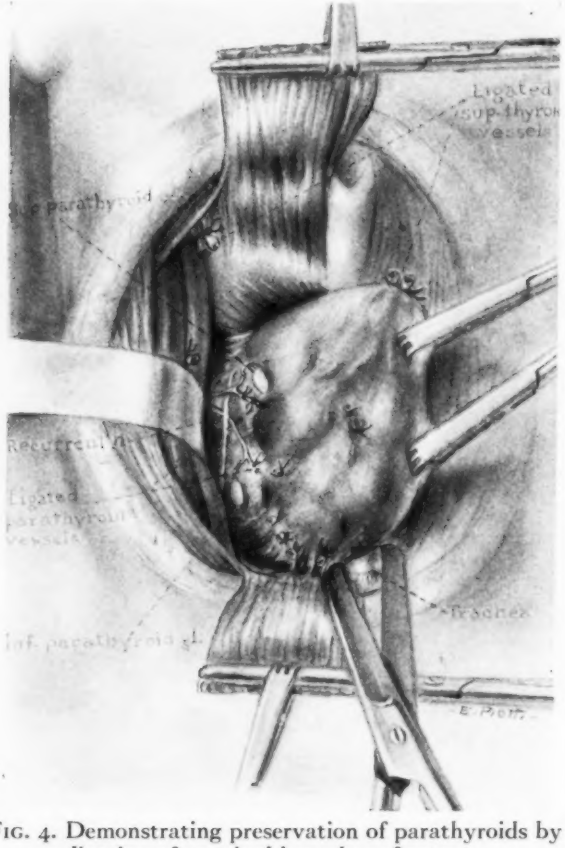

Fig. 4. Demonstrating preservation of parathyroids by ligation of terminal branches of artery.

so perfected since the days of Kocher that it is unnecessary to recite here the technique of the usual subtotal thyroidectomy. But, whereas in the customary thyroid operation the danger zone,[14] the region of the parathyroids and recurrent laryngeal nerves, is left strictly undisturbed, in total ablation of the normal thyroid gland this zone is cautiously, although deliberately, invaded. Although in the usual subtotal thyroidectomy a varying amount of thyroid residue is left behind, not only for the protection of the parathyroid glands and recurrent nerves but also to avoid the development of myxedema, in the present operation complete ablation is effected with the specific intention of creating the low metabolism of myxedema.

The major steps of the operative procedure may be summarized as follows: A modified Kocher collar incision is made slightly above the episternal notch and, when possible, placed in a natural fold of the skin (Fig. 1). The incision should afford ample exposure, as it is not especially necessary to appeal to the aesthetic sense of an invalided cardiac. A full thickness flap is made down to the deep cervical fascia and reflected upwards as high as the superior border of the thyroid cartilage, exposing the pre-thyroid muscles (Fig. 2). The lower flap is not disturbed by dissection. One thereby avoids entering the fascial planes of the superior mediastinum, with its attending risk of hemorrhage from the inferior thyroid venous plexus and possible damage to the pleura. The anterior jugular veins, often considerably engorged in patients with advanced circulatory failure, are carefully ligated before proceeding with the division of the ribbon muscles. Transverse section of the latter (Fig. 2) offers much better exposure for our purposes than lateral retraction and is, therefore, strongly recommended. The divided muscles are retracted widely in opposite directions, bringing into full view both the upper and lower limits of the corresponding lobe of the thyroid gland.

The anterior surface of the exposed portion of the gland is searched carefully for atypically located parathyroids. Cunningham[15] states that "the range of the exceptional positions which the parathyroids may occupy is wide." In the course of several hundred anatomical dissections, I have occasionally isolated a parathyroid body on the anterior aspect of the thyroid gland. Millzner[16] has reported the presence of these glandules in similar aberrant areas. In one of the patients operated upon (R. D.), a parathyroid gland was identified to the left of a pyramidal lobe about 2 cm. below the hyoid bone.

A pyramidal lobe is always sought for and, if found, entirely removed. It should be borne in mind that this elongated process of thyroid tissue is more often present to the left of the median plane and is occasionally duplicated.

The lateral lobe of the thyroid is next partially dislodged from its normal position in the tracheo-esophageal groove by being grasped with two elevating forceps (Fig. 3). With careful blunt dissection, supplemented by gentle digital manipulation, the lateral lobe is divested of its outer fascial envelope. The gland is further mobilized by ligating the middle (lateral) thyroid vein, a routine technical step advocated by Guthrie[17] in subtotal thyroidectomy. The upper pole is then completely freed. Before the vessels are secured with a double ligature of catgut (Fig. 3), the superior pole is inspected in its entire circumference for the presence of parathyroids. It is uncommon, however, to find them in this region. The ligatures should be introduced from within outwards and, according to Nordland,[18] care should be exercised to avoid inclusion of the external laryngeal nerve which supplies the cricothyroid muscle, a weak adductor but a strong tensor of the vocal cords. Crotti[14] does not share this opinion, stating that injury to the superior laryngeal nerve is an uncommon occurrence.

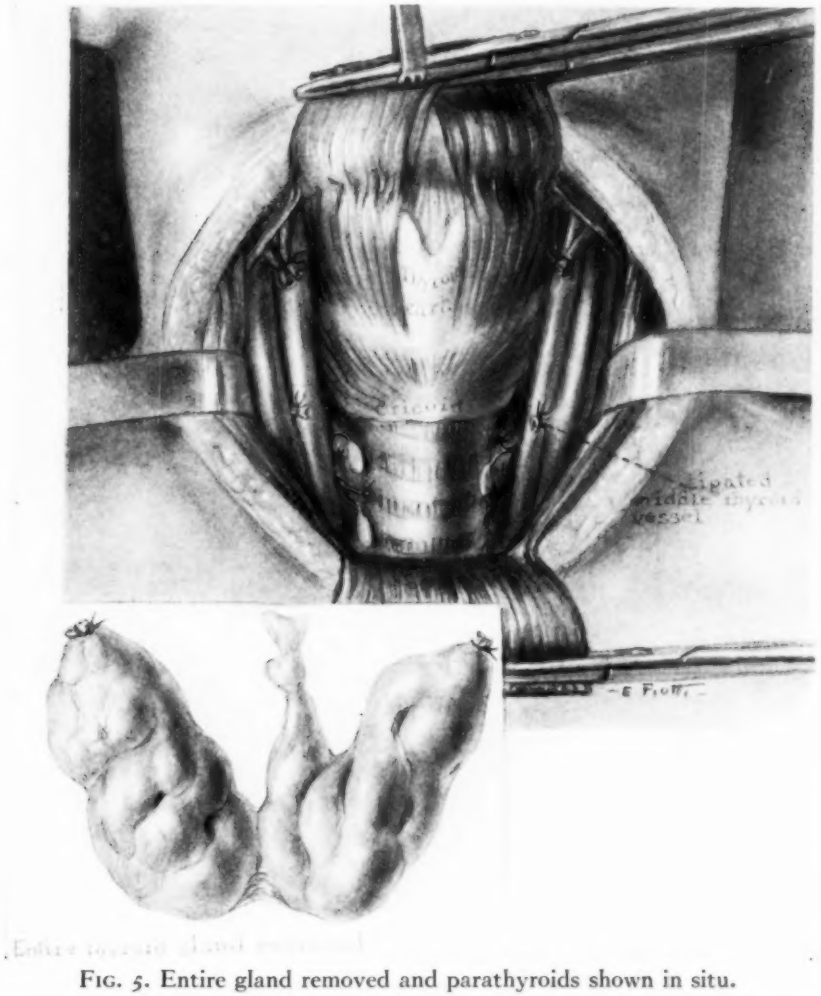

Fig. 5. Entire gland removed and parathyroids shown in situ.

The upper pole detached, one proceeds next to liberate the isthmus from its loose, anterior tracheal attachment by tunneling a curved hemostat beneath it, being careful not to perforate the trachea. With the aid of small, blunt, dissecting scissors, the danger zone of the tracheo-esophageal sulcus is cautiously invaded to identify and isolate the parathyroid glandules (Figs. 3 and 4). They are yellowish-brown, flattened oval bodies about the size of a

lentil or split pea, with a finely granular surface. This granular appearance is of particular aid to the operator in dis-

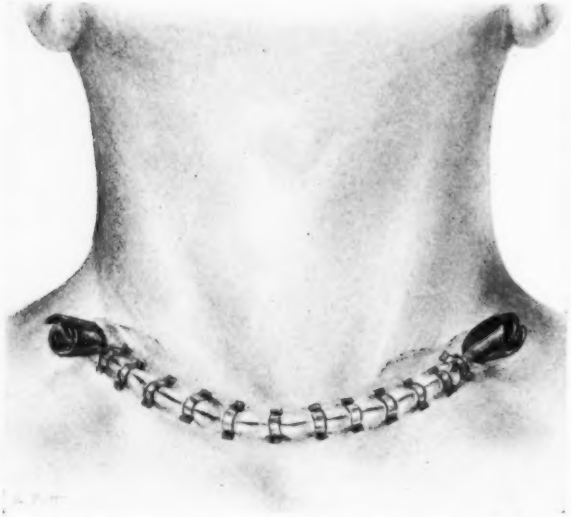

Fig. 6. Closure of wound with drainage, using soft Penrose tubing.

tinguishing them from neighboring lobules of fat. It should not be difficult to differentiate them from the brownish-red, coarser fragments of aberrant or accessory thyroid tissue. They are usually found in relation to the terminal branches of the inferior thyroid artery, each supplied by its own arterial twig. They are most commonly found just medial to the posterolateral border of the lobe. Occasionally, one is found in relation to an independent parathyroid vessel which may arise from the longitudinal anastomotic channel connecting the superior and inferior thyroid arteries. Halstead and Evans[19] suggested this channel of anastomosis as a possible guide to the identification of the parathyroids. While four is given as the usual number, it may be difficult to demonstrate the presence of more than two or even one parathyroid. Competent observers have reported varying numbers, ranging as high as twelve in one cadaver. Because the inferior parathyroid bodies are usually more laterally placed, away from the fibrous tunic of the thyroid gland, and not infrequently supplied and suspended by a rather long arterial twig, their detection and preservation may be more readily effected.

Crile[20] places considerable emphasis on the vulnerability of the recurrent nerves, stating that abductor paralysis is most frequently caused by traction, and that the slightest direct or even indirect pressure on the nerve will interfere with conduction and cause an immediate change in the voice. Judd, New and Mann,[21] however, on the basis of animal experiments, present evidence which indicates that stretching the recurrent laryngeal nerve causes no disturbance in the vocal cords, and that, while pinching may produce a temporary palsy, only ligation or section of the nerve results in permanent vocal cord paralysis.

As the ablation of the gland advances, scrupulous attention is paid to hemostasis. Only the terminal branches of the inferior thyroid artery are ligated in order to conserve the circulation to the parathyroids, although, if necessary, the main trunk of the artery may be ligated. To prevent nerve injury, the intimate anatomic relationship existing between the recurrent laryngeal nerves and the inferior thyroid arteries should be appreciated and respected when ligating the latter vessels near their termination close to the gland. In an earlier publication,[22] it was pointed out that the recurrent laryngeal nerve is more often anterior to the inferior thyroid artery on the right than on the left side of the neck.

After freeing the moulded tracheal surface of the gland from the depth of the tracheo-esophageal groove, one normally encounters, in the final step of the complete separation of the lobe, an adherent zone of attachment of the gland to the trachea just below the arch of the cricoid cartilage (Fig. 3). Crile has called attention to the nerve trunk lying in close contact with the gland at this point, and it strictly behooves the operator to define meticulously and with the least amount of trauma a line of cleavage between the nerve and gland at this very important step in the operation. The fixation of the gland to the trachea in this area may be so firm that

perforation of the trachea conceivably may occur if the utmost care is not taken in peeling off the gland.

If the condition of the patient warrants, and this is usually the case, the entire procedure is repeated on the other side and the gland totally removed in continuity (Fig. 5). A careful search of the entire gland is made for adherent or imbedded parathyroids, which, if found, are reimplanted in the sternomastoid muscle in accordance with the technique adopted by Lahey[22] in the operation of partial thyroidectomy. To avoid the possibility of insult to the laryngeal nerves by the accumulation of serum or the formation of a hematoma, drainage is always instituted. Drains of Penrose tubing are used and made to pass through the sternomastoid muscle on either side. The skin is closed with Michel clips (Fig. 6) and a firm sterile gauze dressing is applied.

CONCLUSIONS AND SUMMARY

1. A new method for the treatment of chronic heart disease is described, this method consisting in the total ablation of the normal thyroid gland in patients without clinical or pathological evidence of thyroid toxicity.

2. The necessity for total ablation of the gland in order to insure a persistently lowered metabolic rate is emphasized.

3. The surgical technique of the operation and the precautions which must be observed are described.

It is with pleasure that I record my thanks to Dr. Charles G. Mixter, Surgeon-in-Chief of the Beth Israel Hospital, for his encouragement and helpfulness.

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[For Remainder of References see p. 209.]

PEPTIC ULCER AND VENOFIBROSIS*

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TO the ever-increasing literature on peptic ulcer we desire to add another chapter. In our studies on venofibrosis, we discovered clinically that the greatest number of cases demonstrating this lesion occurred in patients suffering, or having suffered, from peptic ulcer. It is true that not all had symptoms referable to the gastrointestinal tract. Some had no symptoms whatever. Two had generalized fibrosis with symptoms of portal sclerosis and recent thromboses of that vein. In these cases the abdominal condition was diagnosed from the condition of the superficial veins, biopsy of the arm and leg veins showing lesions of those vessels. Similar lesions have been seen by us in thromboangiitis oblitèrans, and in juvenile arteriosclerosis. The number of cases in women have been few, because we have, up to the present, seen few females with ulcer. But of these, several showed the lesion. Not all cases of ulcer have shown arm vein lesions, possibly because the ulcers have been of a type different from the usual, or that the dissemination of these fibrotic lesions has not involved the arm vessels.

We shall not recount the predisposing factors of ulcer in detail. But there are a few that are accepted by most authorities as playing a greater or lesser role in the etiology of ulcer. It is sufficient to just mention the role of emotional or psychic strain, the use of tobacco, the so-called "ulcer diathesis," the polycythemia, the tendency to low blood pressure, and the small hearts. It is in just this type of individual that venofibrosis is a very characteristic sign. We have seen this fibrosis in hundreds of ulcer cases here and abroad. The veins selected for examination are those about the elbow and ankle. Attempts to withdraw blood from these veins are often futile because of their fibrosed state. In one very marked ulcer case in which operation had been performed, the generalized fibrosis was so extensive that transfusions could not be given in any veins in the body.

Age bears no relation to venofibrosis. The most marked thickening was present in two children with ulcer histories dating back for the greater part of their lives. Demonstration of these veins convinced doubters of the possible intimate association of the two conditions. Recently Cushing in his article on "Peptic Ulcer and Interbrain"[1] called attention to a very significant series of cases and his view confirmed us in the belief of a neurogenic vascular origin of the disease.

Venofibrosis has been a completely overlooked condition. Why the veins, so important in the circulatory system, have been ignored by all students is difficult to understand. The "negative" or "passive" action of these vessels seems to have been deeply impressed upon clinicians, physiologists and pathologists alike. One celebrated cellular pathologist abroad assured one of us (L. H.) with a smile, when he viewed our sections, that he knew nothing of veins. Another stated that they were secondary lesions and closed the subject with a shrug. Many had never seen such changes, though they had studied pathological specimens for years.

A single glance at a section of vein, with its lining endothelium, its various coats, its vasa vasorum, and its intricate nerve supply, must at once call attention to the lack of simplicity and passivity of the veins, in the circulation. A Berlin patholo-

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gist looking at some of our sections declared that the vein showed definite spasm. Strangely enough, this observer has been stressing, as have many others, the role of spasm in arterial disease and yet he ignores its possibility in causing vein changes. Why not assume until definite evidence is available that repeated spasm of the musculature of the veins may cause thickening of the vessel, changes in its wall, and circulatory and nutritional changes in the surrounding tissues? Perhaps the much-discussed hunger pains may be accounted for by vein spasm due to nerve irritation, as well as to muscle contraction and even the usual hyperacidity may be accounted for on the same theory. If this vein fibrosis is widely disseminated, as we have demonstrated it to be in hundreds of cases, Cushing's recent communication certainly adds support to the theory of a neurogenic vascular origin of ulcer. Physiologists have long recognized the existence of a nerve supply to veins as complete as that of the arteries, and others have called attention to the nerve tissue found in the capillary system.

Is there any basis for the support of the theory of venofibrosis being a predisposing factor in the production of peptic ulcer? Analogies are dangerous bases for argument, but surely they are at least as valuable as the evidence of experimental ulcers produced in dozens of ways, and which do not resemble the so-called peptic ulcer in many particulars. These experimental defects are a far cry from the real lesions in human beings.

Varicose ulcers of the leg in many features resemble peptic ulcers. Size, shape, consistency, structure, duration, tendency to heal and re-appear at the old or other regions, and their pathologic pictures seem the nearest approach to the lesions in the digestive tract. The part taken by the vein in the production of the leg ulcer is definite. In the last edition of Kaufmann's Pathology,[3] there is a plate, here reproduced, showing a section of saphenous vein from a patient with varicose ulcer (Fig. 1). He calls the lesion "chronic hyperplastic phlebitis." We prefer the term venofibrosis for reasons given in our original article. That vein conditions in the leg cause spasmodic pains in the muscles is illustrated in daily practice by patients with cramps in the legs due to the veins, long before the ulcer appears.

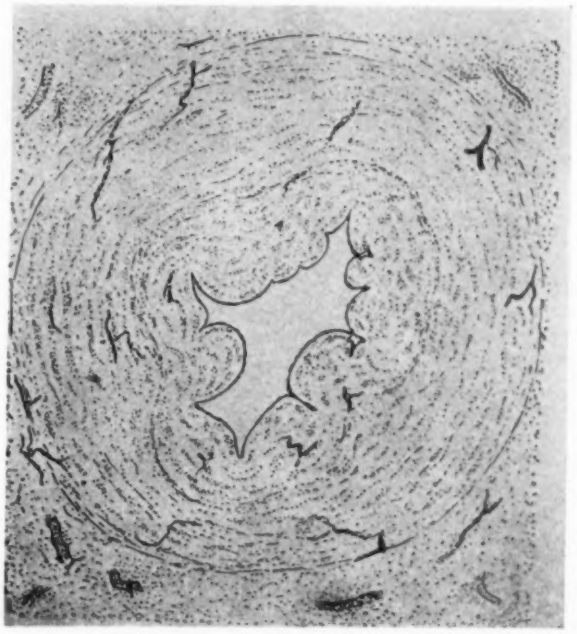

Fig. 1. Chronic hyperplastic phlebitis (Phlebosklerose) of the saphenous vein in patient with crural ulcer. (After Kaufmann.[3])

The presence of these veins does not necessarily indicate that there is ulcer, but where they exist in patients with digestive disturbances, they are a significant addition to the diagnosis of disease in the upper digestive tract. Frequently it has helped in the differential diagnosis in doubtful conditions. We consider the existence of these changed vessels a markedly predisposing factor to ulcer production. Just as ulcer of the leg requires an exciting, immediate cause, so does peptic ulcer need another provocative impetus.

The problem before us, after being convinced of the clinical association of peptic ulcer and venofibrosis, was to determine the condition of the veins in the stomach and duodenum of ulcer patients. Are these fibrous veins potentially existent in the

upper digestive tract, or are they only secondarily involved? With these questions we shall now concern ourselves.

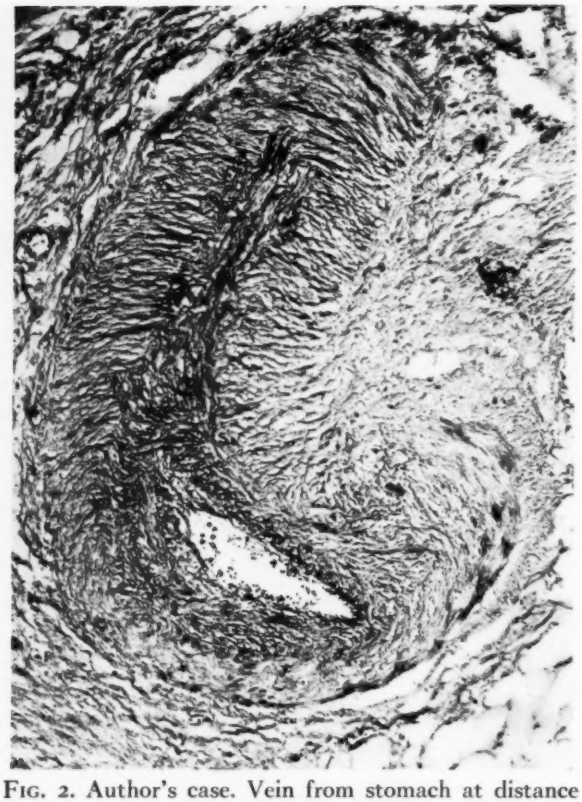

Fig. 2. Author's case. Vein from stomach at distance from ulcer. Note similarity in fibrotic changes to those seen in Figures 1 and 4.

PATHOLOGY

Arteritis with thrombosis is present in many of our cases. Cushing, Stewart and others show it in their illustrations. No mention is made of the condition of the veins except in Reimann's translation of Kaufmann's work.[4] We have studied the veins near the ulcer and at a distance from the ulcer, and in different parts of the body.

In our original article[2] the veins in venofibrosis were described as follows:

The vessels are always small, never dilated. We have never seen any inflammatory changes, such as infiltration, scar formation, oedema, necrosis, etc. Frozen sections have never shown fatty droplets, nor have we ever seen calcium deposits. The histologic changes seem to be uniform and constant. The very earliest changes seem to be the loss of the endothelium and the hyalinization of the denuded surface. There is a marked hyperplasia of fibrous connective tissue particularly in the media, and to a lesser extent in the intima, the latter very

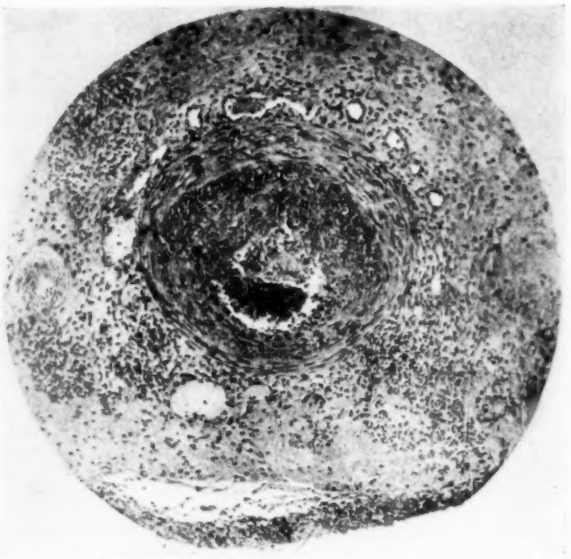

Fig. 3. Vein in wall of gastric ulcer. Underlying inflammation and thrombosis can be recognized definite fibrosis of media and intima. (After Kaufmann.[3])

frequently showing irregular, thickened, projections into the lumen. Often both media and intima are so involved that it is difficult to determine the limitations of the layers. A striking feature is the appearance of small muscle bundles surrounded and separated by enormously thickened connective tissue septa, at times even the individual muscle cells are thus surrounded and separated, the entire media in a preparation stained either by van Gieson's method, or by Mallory's aniline blue method, having a striking alveolar appearance, resembling, architecturally, the isolated appearance of islets of hepatic parenchyma surrounded by connective tissue in Laennec's cirrhosis of the liver.

Most frequently the entire circumference of the vein is involved, but occasionally (especially in advanced cases) there is a very pronounced irregularity of the fibrotic process, so that there seem to be localized bulgings into the lumen, and one part seems to be several times as thick as another (venofibrosis circumscripta s. nodosa). The lesions are equally pronounced in the vasa vasorum.

This description of the veins in the arms and legs of patients with venofibrosis is equally applicable to the veins in the

stomachs of ulcer patients, except for a few minor changes. These changes include edema and inflammatory changes, with occasionally necrosis of the wall. Obviously this is the effect of the presence nearby of an inflammatory process, namely the ulcer. As we get farther away from the ulcer area, less inflammatory changes are found, and at some distance from the necrotic area, the pure venofibrotic lesion is seen, just as it is present in the veins of the extremities.

The argument may be brought forth that the lesion is a secondary one. The inflammatory change is obviously just that. But it is a secondary change superimposed on the originally present venofibrosis, shown by the fact that the veins at some distance from the ulcer show no inflammation, but just the original venofibrosis. (Figs. 2 and 3.)

SUMMARY

We have attempted to prove that peptic ulcer is associated in the majority of cases with venofibrosis, a generalized underlying constitutional condition. The fact that this is not present in the superficial veins of all ulcer patients does not prove its absence from the stomach, just as its presence in the veins of the extremities does not prove its presence in the stomach. The condition is a disseminated one but not a universal one, so that certain groups of the veins may be affected while others are not. However its presence in the superficial veins should lead, at least, to a suspicion that it may also be in the stomach wall. The ulcer itself represents an added, necessarily infective lesion, with the veins as a possible basis. Ulcers of the stomach may be as various as ulcers anywhere. Our observations lead us to believe that the chronic, typical, so-called "peptic ulcer" is almost always associated with venofibrosis. This fact affords the physician what may be a valuable aid in diagnosis, namely, a superficial presumptive sign of ulcer. As a practical application of this theory it would be well to consider patients with superficial venofibrosis as potential ulcer patients, so that definite precautionary measures (dietetic and others) may be instituted to prevent the occurrence of the ulcer; or to facilitate recovery when one is present in a comparatively mild form.

Fig. 4. Venofibrosis. Arm vein in patient with gastric ulcer. Note marked thickening and fibrosis of intima and media.

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EFFECT OF FOODS ON POSTOPERATIVE DISTENTION

AN EXPERIMENTAL STUDY*

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I. INTRODUCTION

THERE are a number of well-known mechanisms for the accumulation of gases in the intestine, such as swallowed air, the diffusion of gases from the blood, and the elaboration of gases by foods interacting with intestinal contents. McIver, Benedict and Cline[1] recently presented evidence to show that a major cause of postoperative distention was swallowed air. In this report we shall emphasize, on the basis of experimental evidence, the significant role which certain foods, commonly administered to patients in the immediate postoperative period, may play in distention. It is hoped that the data offered may lead to a more effective control of this distressing aspect of the postoperative convalescence.

The only gases of importance in distention are those which are not rapidly absorbed. It has been well established through the work of Schoen[2] and McIver, Redfield and Benedict[3] that these are H_2, CH_4, and N_2. Their disposal for the most part requires expulsion by peristalsis. A small amount of CO_2 is always found in the gas content of the gut, but, in general, the readily absorbable gases (CO_2, H_2S and, to a much smaller degree, O_2) do not become significant distending agents until their rate of accumulation exceeds their absorbability. This may occur in the colon with mild or moderate degrees of venous obstruction, but Kato[4] and Schoen[2] have pointed out that in the small intestine only extreme degrees of venous obstruction will noticeably decrease the efficiency of gas absorption.

When the gastrointestinal tract is structurally intact the colon is not a serious factor in postoperative distention, since by a rectal tube or enema this portion of the gut is readily decompressed. For this reason our experiments were confined to the stomach and small intestine. Except where specifically stated, we did not interfere with the circulation of the gut, so that we were not dealing with distention attributable to absorbable gases, at least not until the distention had reached a degree sufficient to compress the veins in the gut wall.

II. PROCEDURE

A. CONTROL OBSERVATIONS:

1. *Volume of Gas in the Structurally Intact Small Intestine of the Starving Cat Postoperatively.* Basal conditions for a quantitative study of the effect of foods on distention required, first, the determination of the amount of gas ordinarily to be found in the small intestine after a measured postoperative period during which no food is allowed. Cats, which were allowed water but no food for thirty hours, were subjected to laparotomy with the usual sterile precautions. The intra-abdominal manipulation consisted of only that amount necessary to find and deliver to vision the ileocecal valve. The wound was closed and the animals were allowed only water. After stated intervals they were electrocuted and the amount of gas in the small intestine was measured by aspirating the entire contents into a large leak-proof syringe.

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Table I
SIMPLE LAPAROTOMY WITH SLIGHT BOWEL MANIPULATION

Experiment Number	*Postoperative Starvation Period (Hours)*	*Gas in Small Intestine (C.c.)*	*Fluid in Small Intestine (C.c.)*	*Anesthesia**
1	41	9	4	Ether
2	44	1	0.5	Ether
3	46	0.5	3.5	Ether
4	67	3	2	Ether
5	42	1	3	Ether
6	45	2	3	Ether
7	43	0	5	Nembutal intraperitoneally
8	42	1.5	5	Nembutal intraperitoneally
9	43	7	2	Nembutal intraperitoneally
10	42	3.5	5	Nembutal intraperitoneally
11	41	1	4	Nembutal intraperitoneally

* For comparison of the effect of the type of anesthesia six animals were operated under ether, and five under nembutal intraperitoneally (0.4 c.c. per kg. body wt.). The difference in resulting gas volumes is nil. All subsequent operations were done with nembutal.

Table I shows that the total amount of gas after a period of roughly forty-four hours is negligible, averaging about 2.5 c.c. Presumably air has been swallowed and gases have formed locally, but peristalsis has been sufficiently effective to avoid distention by the relatively non-absorbable gases.

2. *Volume of Gas in the Obstructed Small Intestine of the Starving Cat.* It is of course possible that the amount of operative trauma in the foregoing series of cats may have been inadequate to induce an effective functional ileus, the usually accepted cause of postoperative distention. But, as will be shown below, operative trauma to the gut of the cat, however severe, which still leaves it structurally intact, is not capable of causing appreciable distention. Failing in our effort to produce a satisfactory degree of functional ileus, we were obliged to utilize a simple mechanical ileus as a substitute in order to find out how much non-absorbable gas might have collected had we been able to establish an adequate degree of functional ileus. Accordingly, the experiments were repeated with this addition, that when the ileocecal valve was delivered to view, a ligature was placed around it and tied tightly, so as to completely obstruct it.

Table II
LAPAROTOMY WITH ILEOCECAL VALVE TIED; NEMBUTAL ANESTHESIA

Experiment Number	*Postoperative Starvation Period (Hours)*	*Gas in Small Intestine (C.c.)*	*Fluid in Small Intestine (C.c.)*
1	42	11	32
2	42	43	11
3	44	3	17
4	47	4	9
5	44	64	24
6	42	10	70

The results are given in Table II. The wide variation in volumes of gas is notable. Distention occurred in only 2 out of 6 instances after a period of about forty-four hours. Figure 1 shows the *x*-ray appearance of the abdomen of two cats treated as just described, one of which had distended coils of small intestine with a gas volume of 54 c.c., while the stomach contained 46 c.c. (total 100 c.c.). The other had collapsed coils with a gas volume of only 6 c.c. Such variability can, in accordance with McIver's thesis and with that of clinical experience, be explained on the basis of the varying degrees of aerophagia in which different individuals will indulge.

Since these animals were offered water postoperatively, it may be that those which drank also gulped air in corresponding volume. Because of its clinical interest, we proceeded to inquire whether the total withdrawal of water by mouth, to suppress air swallowing as far as possible, would help to counteract distention. The experiments listed in Table II were repeated and no food or water was allowed postoperatively. After similar periods the total gas volume was determined. Table III shows that air may be swallowed* in considerable volume whether or not liquid is taken, and that the gas volume is again widely variable.

*That the gas is swallowed air will appear from subsequent data.

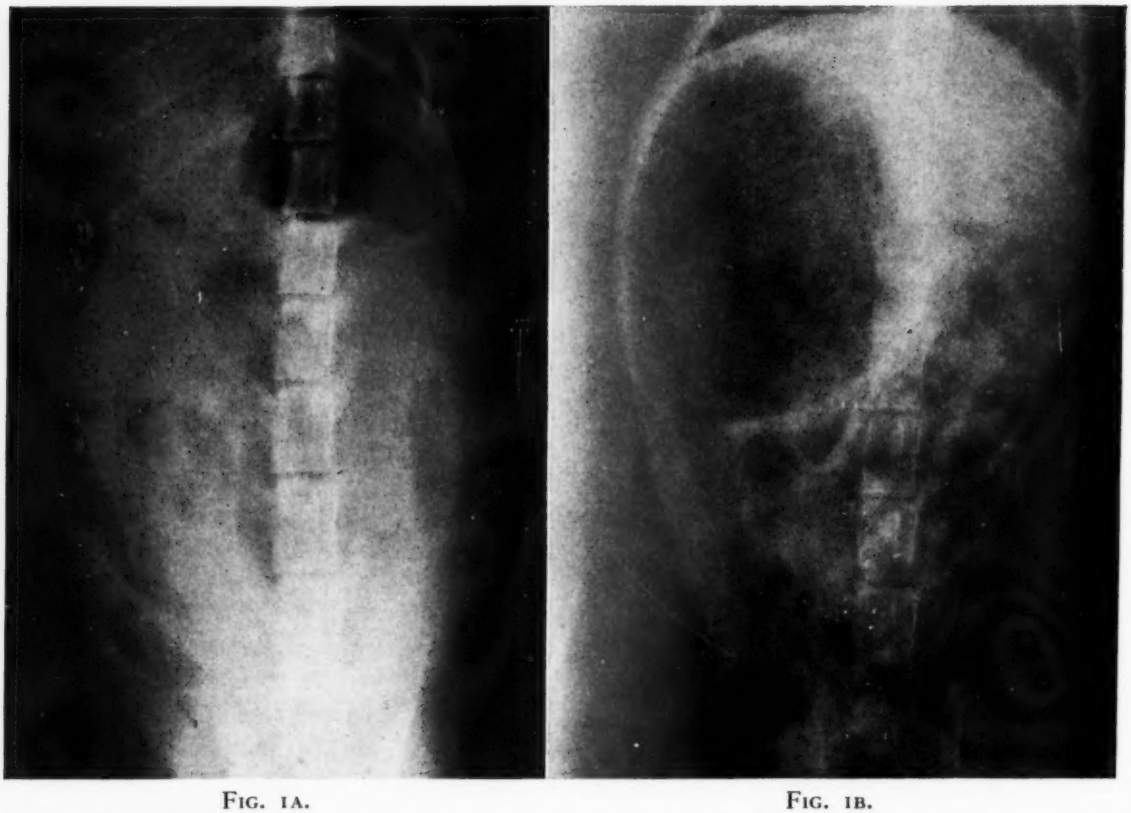

FIG. 1A. FIG. 1B.

FIG. 1. A. *X*-ray appearance of intestines of a starving cat forty-four hours after ligation of ileocecal valve. Gas content of small intestine 6 c.c. B. Greatly distended coils of another cat treated exactly the same way after the same interval, with gas volume of 54 c.c. in small intestine and 46 c.c. in stomach (total 100 c.c.).

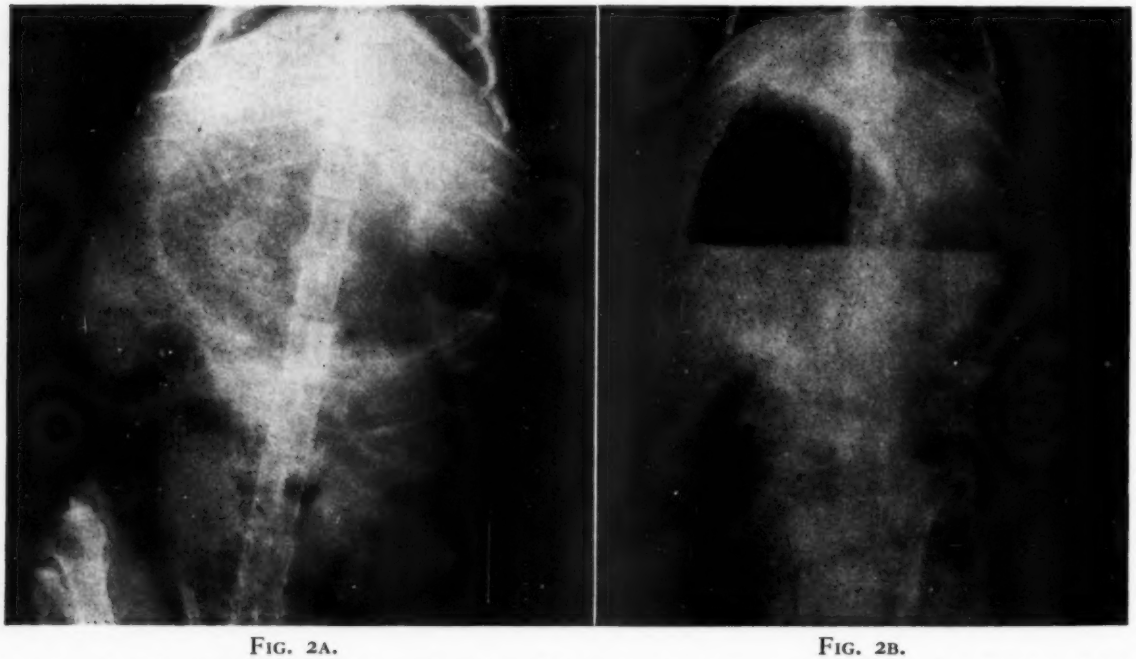

FIG. 2A. FIG. 2B.

FIG. 2. A. and B. *X*-ray appearance of stomach and small intestine of cat twenty-four hours after ligation of cardia and ileocecal valve and injection of 100 c.c. of soft custard into stomach. A. Prone position. B. Vertical position. Total gas volume 210 c.c.

Table III
ILEOCECAL OBSTRUCTION; NO FOOD OR WATER POSTOPERATIVELY

Experiment Number	*Postoperative Starvation Period (Hours)*	*Gas in Small Intestine (C.c.)*	*Fluid in Small Intestine (C.c.)*
1	44	44	40
2	43	9	18
3	42	24	85
4	42	22	40

3. *Effect of Trauma to the Intestine on the Degree of Postoperative Distention in the Starving Cat.* Although an effort was made to keep the degree of operative manipulation in the preceding experiments as uniform as possible, we next determined the extent to which trauma to the gastrointestinal tract contributed to the degree of postoperative meteorism. There is a clinical impression that such a relationship exists, presumably on account of the induced adynamic ileus. Although one cannot accurately quantitate the relationship between the amount of trauma and the degree of distention, a rough approximation is possible by comparing the amount of gas accumulating above an ileocecal tie in animals treated exactly alike, except that some were subjected to minimal handling ("slight trauma," Table IV) incident to placing the tie, others to moderate evisceration ("moderate trauma") and a few others to complete evisceration of the gut plus strong traction on the mesentery ("severe trauma").

The results give no evidence to the effect that handling the intestines produces a proportional degree of meteorism. The volume of gas present in the animals with minimal trauma was as great as (if not greater than) in those with moderate or severe trauma for a corresponding postoperative period. *X*-ray studies at twenty-four-hour intervals for three days on other animals with similar gradations of trauma, but without obstruction, revealed no essential variation in the gas content of the gastrointestinal tract.

These observations are in accord with the clinical experience that the incidence of distention in operations on the gastrointestinal tract is less than in operations on, for example, the pelvic organs or the biliary system (Cutting,[5] McIver[1]). This may in part be attributable to the usual avoidance of peroral feeding in the former for the first few days after operation. Another factor is the probability of a more lasting inhibition of motor activity and a more intense reversal of the gradient of irritability (Alvarez[6]), such that any swallowed air or food would be less likely to gain access to the small intestine than would be the case in operations not directly involving the gastrointestinal tract.

Table IV
ILEOCECAL OBSTRUCTION; VARYING DEGREES OF PURPOSEFUL INTESTINAL TRAUMA

Experiment Number	*Slight Trauma: Gas in Small Intestine (C.c.)*	*Moderate Trauma: Gas in Small Intestine (C.c.)*	*Severe Trauma: Gas in Small Intestine (C.c.)*	*Postoperative Period (Hours)*	*Clinical Appearance before Electrocution*
1	29			23	Good
2	27			27	Good
3	58			28	Good
4	2			27	Good
5	54			48	Fair
6		8		18	Bad
7		56		90	Good
8			12	27	Good
9			0	28	Bad
10			54	48	Poor
11			47	72	Fair
12			9	48–66	Found dead

In the latter operations the gradient is possibly disturbed to a lesser degree, so that swallowed air or non-absorbable gases formed locally may be propelled (together with fluids), however inefficiently, along the gut, but with distention resulting because peristaltic activity is not yet sufficiently well coordinated to advance the intestinal contents rapidly and effectively.

4. *Extent of Local Gas Formation in the Stomach and Small Intestine of the Starving Cat.* The preceding experimental data were presented with the assumption that most of the gas in those experiments was of oral origin. That this assumption is justified is shown by the next group of experiments

in which, in addition to ligation of the ileocecal valve, the esophagus in the neck was tied off. By comparing the figures for gas volume in Table v with those of Tables II and III it will be obvious that the gases retained in the latter were indeed, for the most part, of oral origin.

TABLE V
SIMULTANEOUS LIGATION OF ESOPHAGUS AND ILEOCECAL VALVE

Experiment Number	*Postoperative Starvation Period (Hours)*	*Gas in Small Intestine (C.c.)*	*Fluid in Small Intestine (C.c.)*
1	41	8	33
2	44	0	0
3	43	12	8
4	41	5	2

Even the small amount of gas that was recovered in the experiments of Table v may have been swallowed air, for when the esophageal ligation was performed twenty-four hours before the ileocecal ligation, to allow the expulsion of any air present at the outset, the gas was reduced to zero in three instances, and to 3 c.c. in one animal carried for ninety-six hours (see Table VI). The postoperative periods were sufficiently prolonged to exclude the objection that time enough for local gas-producing processes to act was not allowed.

TABLE VI
LIGATION OF ESOPHAGUS FOLLOWED IN TWENTY-FOUR HOURS BY ILEOCECAL LIGATION

Experiment Number	*Hours after Ileocecal Ligation*	*Gas in Small Intestine (C.c.)*	*Fluid in Small Intestine (C.c.)*
1	26	0	3
2	26	0	1
3	49	0	5
4	73	3	10

Moreover, the insignificance of local gas formation obtains whether the circulation to the small intestine is intact or not. In 8 cats in which the entire small intestine was rendered gangrenous, or nearly so, by ligation of the superior mesenteric artery, we found no distention. The amount of gas averaged 4.5 c.c. This confirms the observations of Scott *et al*[7] who found the same true for strangulated loops of varying lengths.

The conclusion is warranted that in a starving animal gases generated by local processes are unimportant in the production of meteorism in the first few days following a laparotomy, and that gaseous distention, when it does occur during starvation, whether because of functional or mechanical ileus, is due to swallowed air.

As a preliminary to observations on gas production by foods when placed in contact with different segments of the stomach and small intestine, closed empty loops of different lengths and location were created to make certain that no vital difference in local gas formation, little as it is, exists between various portions of the empty intestinal tract (exclusive of the colon). Table VII shows that a significant difference is not recognizable, although exclusion of the ileum from the loop resulted in no gas whatever on the two occasions it was tried.

TABLE VII
LOCAL GAS FORMATION IN VARIOUS EMPTY LOOP SEGMENTS OF GUT

Experiment Number	*Type of Loop*	*Age of Loop (Hours)*	*Gas in Loop (C.c.)*	*Fluid in Loop (C.c.)*
1	Cardia to ileocecal valve	24	7	7
2	Cardia to ileocecal valve	24	12	11
3	Cardia to middle of small intestine	24	0	75
4	Cardia to middle of small intestine	24	0	8
5	Pylorus to ileocecal valve	41	10	20
6	Pylorus to ileocecal valve	45	18	108
7	Last foot of ileum	24	1	6

Before eliminating local gas formation as a contributory source of distention in these animals, it should be pointed out that 7 out of the 8 animals of Tables v and VI had so little fluid in the gut that fermentation or putrefaction may have thereby been inhibited. These animals were, in spite of

the fact that they received 50 c.c. of 2.5 per cent glucose in saline infusions daily, probably seriously dehydrated owing to the loss of large quantities of saliva, the secretion of which seemed to have been accelerated by the irritation of the ligature in the neck. To avoid this source of dehydration a further series of cats was subjected to simultaneous ligation of the cardia and ileocecal valve. Any gas present at the time of ligation was aspirated from the involved portion of the gut, though ordinarily the stomach and small intestine were found empty and collapsed. As will be observed from Table VIII, the gas and fluid volumes remain about the same as those of Table V.

TABLE VIII
LIGATION OF THE CARDIA AND ILEOCECAL VALVE

Experiment Number	*Postoperative Starvation Period (Hours)*	*Gas in Small Intestine (C.c.)*	*Fluid in Small Intestine (C.c.)*
1	20	1	5
2	24	6	2
3	23	9	3
4	96	0	3

It appears, therefore, that the low fluid volume is not to be attributed to excessive dehydration, but to the absence of gas.* It is at the same time evident that fluids in the obstructed intestine of a starving cat in the absence of swallowed air will not reach a sufficient bulk to act by themselves as an agent for the further secretion of fluids. From the foregoing observations it can be stated (1) that the primary agent for increasing intraluminary tension in simple obstruction in a starving animal seems to be gas and not fluid; (2) that gas is therefore the initial stimulus for the secretion of fluids under such circumstances. The lack of parallelism between the volumes of gas and fluid in simple obstruction (see Tables II and III) need not be surprising because the tension increase responsible for further fluid secretion depends not alone on gas volume, but also on muscle tone.

It will be shown from subsequent data that the tension of fluids cannot *per se* act as an agent for the accumulation of gas. This is not to deny that fluids, once they have been secreted in quantity, may facilitate local gas production by fermentation or putrefaction. But such fluids, unless they contain nutriment of extrinsic origin, will not be in themselves sources of gas, since they consist merely of water, mucus, salts and enzymes.

B. Effect of Foods on Gas Production:

The foregoing data indicated the necessity of excluding swallowed air (by ligation of the cardia) in order to quantitate the non-absorbable gas forming capacity of foods. Collection of these gases required, as in the control experiments, the use of an obstructing ligature. This amounts to the use of a closed loop. The type most often utilized extended from the cardia to the ileocecal valve or to a variable distance proximal to it. The food was injected, as a rule, by needle and syringe into the stomach, and the gas and fluid volumes were measured quantitatively eighteen to twenty-four hours later unless otherwise stated. The general technique of the procedure was in other respects the same as in the control experiments.

The foods selected for study consisted for the most part of a variety commonly administered during the liquid diet stage of the postoperative convalescence, the period when gaseous distention is ordinarily at its maximum. It will be most convenient to tabulate and discuss the data for the various classes of foods, i.e. carbohydrates, proteins, fats and mixed types, separately and then summarize the findings with a general comment on the indications for clinical application which they suggest.

1. *Carbohydrate Foods* (*Table* XII). Since the commonest type of immediate postoperative alimentation consists of liquid

* Further support to this statement appears in Table III. In this group of cats water deprivation was just as complete as in those of Table VIII, but since air could be swallowed, the fluid volumes were considerable.

carbohydrates, we made a rather extended study of them. By way of orientation for observation of their effects in the animal, an *in vitro* experiment was carried out in which varying concentrations of glucose solutions and other substances were separately inoculated from the same sample of small intestinal contents of a starved cat, and incubated for twenty hours at 37.0°C. Physiological saline, used as the diluent for the glucose solutions, served as their culture medium, while the other foods (liquids) were cultured as such. A similar series was run with the addition of powdered peptone and beef extract ("Difco") to make the medium more or less equivalent to nutrient broth. Both series were run in duplicate with a light and a heavy inoculation. All the tubes were buffered with a small piece of autoclaved intestine. Where gas was produced, the results were proportional to the strength of the inoculation, while the nutrient broth tubes usually gave slightly larger amounts of gas than those without it. Since the results were qualitatively the same for each food in every series, only the figures for the heavy inoculation in nutrient broth are recorded, expressed in percentage of total gas tube length, as follows:

Table IX

50 per cent glucose	0
25 per cent glucose	trace
10 per cent glucose	20 per cent
5 per cent glucose	20 per cent
2 per cent glucose	30 per cent
Water	0
Nutrient broth	trace
Milk	15 per cent
Orange juice	0
Bean puree	5 per cent

Apparently, then, *in vitro* 25–50 per cent glucose solutions inhibit gas formation, while higher dilutions do not. (The reason for the absence of gas in orange juice (a weak solution of glucose) is not clear.) It can be assumed therefore that strong glucose solutions must become sufficiently dilute before they can act as a source of gas, and that the period preceding complete absorption must be long enough to permit gas production by bacterial activity.

With this in mind we created stomach loops by tying the cardia and pylorus, and injected 20 c.c. of 50 per cent glucose into each of five loops so prepared. At the end of twenty-four hours the gas and fluid volumes were determined. Since the stomach cannot absorb glucose, the fluid volumes should represent true dilution values and one should expect gas formation in proportion to the degree of dilution.

The following are the results (Table x):

Table X

Exp. No.	*C.c. Gas*	*C.c. Fluid*	*Calculated Final Concentration of Glucose, Per Cent*
1	0	78 (syrupy)	12.8
2	0	90 (syrupy)	11.1
3	24	145	6.9
4	30	120	8.3
5	270	207	4.8
6*	2	114	8.7

* Loop of jejunum.

These figures suggest that strong glucose solutions may or may not produce gas in the gastrointestinal tract according to the degree of final dilution, and that a concentration in the vicinity of 5 per cent will result in extensive gas formation if absorption is delayed. Glucose in this concentration (which is isotonic with the blood) should however be so rapidly absorbed in the healthy intestine as to allow little or no time for bacterial action.

But several contingencies will delay absorption and facilitate gas formation even providing the glucose is sufficiently dilute to begin with: (1) failure of the glucose to reach the ileum where absorption is most efficient; (2) too great a quantity to be promptly absorbed; and (3) impaired absorption efficiency, e.g. because of a vascular impairment of the gut wall.

In this connection the following corroborative data are offered. Closed ileal loops, with or without vascular impairment, were fashioned, containing either 50 per cent or 5 per cent glucose solutions in amounts just sufficient to fill the loops without

tension. At the end of twenty-four hours the gas and fluid were measured, and the loop contents in several instances analyzed for glucose with the results listed in Table XI.

These figures show that in the ileum, where the absorption of glucose is presumably most efficient, 5 per cent glucose will not produce gas unless absorption is it does not, as one should expect, absorb 50 per cent glucose as well as a similarly embarrassed loop does a 5 per cent solution (compare e.g. Exp. 12 and 13 with 5 and 6); and there is therefore more gas to be found in the former case under such circumstances. It would seem in general that in the healthy intestine one should find greater gas formation by glucose solutions stronger than 5

TABLE XI

GAS PRODUCTION IN LOOPS OF ILEUM BY GLUCOSE

Experiment Number	*Glucose Injected (C.c.)*	*Gas after 24 Hrs. (C.c.)*	*Fluid after 24 Hrs. (C.c.)*	*Glucose in Fluid (Per Cent)*	*Condition of Loop at Time of Injection of Glucose*
A. 5 PER CENT GLUCOSE					
1	16	0	1	0	Short fresh loop; circulation intact.
2	7	0	0	0	Short fresh loop; circulation intact.
3	45	0	2	0	Long 48 hr. old empty loop; slight congestion and edema at time of injection of glucose.
4	18	9	60	0	24 hr. old obstructed loop containing food, emptied and then injected with glucose.
5	18	4	3	0	24 hr. old obstructed loop containing food, emptied and then injected with glucose.
6	15	12	0	0	24 hr. old obstructed loop containing food, emptied and then injected with glucose.
7	8	60	4 (bloody)	0	Short fresh loop; all veins tied off.
8	11	18	10 (bloody)	0.02	24 hr. old obstructed loop; all vessels tied off.
9	40	42	18	1.18	24 hr. old obstructed loop; Animal died a few hours postop. These figures like the rest were taken after 24 hours. Equivalent to a purposefully created dead loop of th duration.
B. 50 PER CENT GLUCOSE					
10	15	2	0	0	24 hr. old obstructed short loop containing food, emptied and then injected with glucose.
11	15	2	20	?	24 hr. old obstructed short loop containing food, emptied and then injected with glucose.
12	10	6? (Some lost)	20	0.62	24 hr. old obstructed short loop containing food, emptied and then injected with glucose. Peritonitis.
13	24	58	50 (chiefly mucus)	1.16	24 hr. old obstructed long loop containing food, emptied and then injected with glucose.

delayed either on account of a gut wall injured by a preceding period of obstruction or by deliberate vascular injury, the amount of gas formed being in proportion to the degree of impairment. Reference to the column labelled "Remarks" will show that Experiments 1–9 have been listed in a sequence corresponding to the increasing degree of loop injury, and that the gas formation in general rises parallel with this increase.

Similarly, in the case of 50 per cent glucose, much or little gas will form according to the speed of absorption. It is remarkable that a short loop of ileum in good condition can so well dispose of such relatively huge amounts of glucose. When the loop is partly embarrassed, however, per cent because of the longer sojourn required by the period of dilution to the isotonic level (about 5 per cent). Subsequent data will bear out this assertion.

With these preliminary remarks we may proceed with a consideration of the main body of observations on carbohydrate solutions, listed in Table XII.

By way of clarification a few words are necessary regarding various features of this table as well as Table XIII. For a proper appreciation of the significance of the figures for gas volume, we refer again to Figure 1 (B) in which a 100 c.c. volume of gas in the stomach and small intestine is shown producing an obvious dilatation. This amount may be taken as an arbitrary average standard for gaseous distention of

Table XII

GAS PRODUCTION IN CLOSED LOOPS BY CARBOHYDRATE FOODS

Exp. No.	Wt. of Cat (Kilos)	Type of Food	Amount of Food (C.c.)	Type of Loop	Gas in Loop (C.c)	Fluid in Loop (C.c.)	Total Gas and Fluid (C.c.)	Condition of Animal before Autopsy	Remarks
1	2.4	Glucose (2 per cent)	40	Cardia to ½ distance from ileocecal valve	2	18	20	Good	
2	1.6	Glucose (2 per cent)	40	Cardia to ¾ distance from ileocecal valve	14	51	65	Good	
3	3.5	Glucose (5 per cent)	40	Cardia to ⅓ distance from ileocecal valve	6	10	16	Good	
4	3.2	Glucose (5 per cent)	40	Cardia to ½ distance from ileocecal valve	9	9	18	Good	
5	2.5	Glucose (5 per cent)	40	Cardia to ¾ distance from ileocecal valve	8	2	16	Good	
5A	2.5	Glucose (5 per cent)	80	Cardia to ¾ distance from ileocecal valve	90	20	110	Good	
6	2.7	Glucose (10 per cent)	100	Cardia to ileocecal valve	188	140	328	Good	
7	3.6	Glucose (10 per cent)	100	Cardia to ⅓ distance from ileocecal valve	20	30	54	Good	
7A	3.6	Glucose (10 per cent)	100	Cardia to ¾ distance from ileocecal valve	101	220	321	Died 20 hrs. postop.	
8	3.4	Glucose (10 per cent)	40	Cardia to ½ distance from ileocecal valve	12	18	30	Good	
9	3.9	Glucose (10 per cent)	40	Cardia to ⅓ distance from ileocecal valve	50	34	84	Good	
9A	3.9	Glucose (10 per cent)	95	Cardia to ⅓ distance from ileocecal valve	90	120	210	Good	
10	2.7	Glucose (50 per cent)	20	Cardia to ileocecal valve	78	7	85	Good	
11	3.6	Glucose (50 per cent)	20	Cardia to ½ distance from ileocecal valve	94	99	193	Good	
12	2.5	Glucose (50 per cent)	40	Cardia to ileocecal valve	241	54	295	Good	
13	3.4	Glucose (50 per cent)	40	Cardia to ⅓ distance from ileocecal valve	115	55	170	Very sick	
14	?	Glucose (50 per cent)	40	Cardia to ½ distance from ileocecal valve	206	135	341	Poor	
15	3.0	Glucose (50 per cent)	40	Cardia to ½ distance from ileocecal valve	291	84	375	Found dead 16 hrs. postop.	Some post-mortem gas formation.
16	?	Glucose (50 per cent)	40	Cardia to ¾ distance from ileocecal valve	170	250	420	Found dead 16 hrs. postop.	
26C	2.3	Glucose (50 per cent)	30	Cardia to ¾ distance from ileocecal valve	2	130	132	Moribund	Fluid seemed syrupy like strong glucose unchanged. Peritonitis.
17	2.0	Cane sugar (50 per cent)	40	Cardia to ileocecal valve	40	165	205	Very sick	Fluid at autopsy contained 0.58 per cent dextrose, 2.1 per cent sucrose.
17A	2.0	Cane sugar (50 per cent)	40	Cardia to ileocecal valve	7	41	48	Died; postop. survival unknown	
18	5.0	Cane sugar (50 per cent)	90	Cardia to ileocecal valve	295	455	750	Good	
19	3.2	Cane sugar (50 per cent)	90	Cardia to ileocecal valve	20	280	300	Fair	Most of fluid was in stomach; probably not converted to dextrose.
78	2.2	Cane sugar (50 per cent)	60	Cardia to ileocecal valve	280	150	430	Found dead 16 hrs. postop.	Had some undigested meat, eaten 6 hrs. preoperative, in stomach.
20	2.6	Grape juice { 17 per cent glucose / 1 per cent sucrose }	60	Cardia to ileocecal valve	34	125	159	Very sick	No reaction to grape juice
21	3.7	Grape juice { 17 per cent glucose / 1 per cent sucrose }	40	Cardia to 8 inches from ileocecal valve	13	22	35	Fair	Incomplete reaction to grape juice.
22	3.5	Grape juice { 17 per cent glucose / 1 per cent sucrose }	40	Cardia to ⅓ distance from ileocecal valve	354	90	44	Fair	No grape juice in recovered fluid.
23	2.5	Grape juice { 17 per cent glucose / 1 per cent sucrose }	40	Cardia to ¾ distance from ileocecal valve	231	198	428	Fair	No grape juice in recovered fluid.
63A	4.1	Grape juice { 17 per cent glucose / 1 per cent sucrose }	90	Cardia to ileocecal valve	3	65	68	Good	{ Dilute grape juice in recovered fluid contains no glucose and 0.25 per cent sucrose.

24	2.4	Orange juice { 3.4 per cent glucose, 1.7 per cent sucrose	40	Cardia to ileocecal valve	14	19	31	Good	
25	2.3	Orange juice { 3.4 per cent glucose, 1.7 per cent sucrose	60	Cardia to ileocecal valve	64	6	70	Poor	
26	2.0	Orange juice { 3.4 per cent glucose, 1.7 per cent sucrose	100	Cardia to ileocecal valve	190	?	?	Good	Peritonitis.
25A	2.3	Orange juice { 3.4 per cent glucose, 1.7 per cent sucrose	110	Cardia to ileocecal valve	255	140	395	Died 20 hrs. postop.	Peritonitis.
27	3.4	Orange juice { 3.4 per cent glucose, 1.7 per cent sucrose	60	Cardia to ileocecal valve	20	0	20	Fair	
27A	3.4	Orange juice { 3.4 per cent glucose, 1.7 per cent sucrose	150	Cardia to 1 foot from ileocecal valve	380	145	525	Found dead 20 hrs. postop.	Recovered fluid had 19 per cent dextrose.
28	3.6	Orange juice { 3.4 per cent glucose, 1.7 per cent sucrose	40	Cardia to ½ distance from ileocecal valve	42	115	157	Fair	
29	4.9	Orange juice { 3.4 per cent glucose, 1.7 per cent sucrose	40	Cardia to ½ distance from ileocecal valve	148	98	246	Fair	
30	2.8	Orange juice { 3.4 per cent glucose, 1.7 per cent sucrose	60	Cardia to ⅗ distance from ileocecal valve	24	?	?	Poor	
30A	2.8	Orange juice { 3.4 per cent glucose, 1.7 per cent sucrose	60	Cardia to ⅗ distance from ileocecal valve	16	?	?	Poor	
31	2.5	Ginger ale { 1 per cent glucose, 3.8 per cent sucrose	100	Cardia to ileocecal valve	266	58	324	Good	
31A	2.5	Ginger ale { 1 per cent glucose, 3.8 per cent sucrose	80	Cardia to ⅗ distance from ileocecal valve	126	80	206	Moribund	
32	2.8	Ginger ale { 1 per cent glucose, 3.8 per cent sucrose	90	Cardia to ileocecal valve	68	31	99	Very sick	
32A	2.8	Ginger ale { 1 per cent glucose, 3.8 per cent sucrose	90	Cardia to ileocecal valve	190	80	270	Found dead 16 hrs. postop.	Recovered fluid contained no glucose.
33	4.5	Ginger ale { 1 per cent glucose, 3.8 per cent sucrose	90	Cardia to ileocecal valve	20	60	80	Good	Recovered fluid contained 3 per cent glucose, 0.5 per cent sucrose.
33A	4.5	Ginger ale { 1 per cent glucose, 3.8 per cent sucrose	150	Cardia to 1 foot from ileocecal valve	37	74	111	Poor	Recovered fluid contained 2 per cent glucose, 0.7 per cent sucrose.
5B	2.5	Canned peach syrup	40	Cardia to ⅗ distance from ileocecal valve	74	10	84	Good	Closed ileal loop under great tension produced for other purposes.
30B	2.8	Canned pineapple juice { 5 per cent glucose, 2 per cent sucrose	60	Cardia to ⅗ distance from ileocecal valve	12	94	106	Moribund	Peritonitis. Fluid looked like diluted syrup.
41A	2.5	Canned pineapple juice { 5 per cent glucose, 2 per cent sucrose	90	Cardia to ileocecal valve	250	105	375	Found dead 16 hrs. postop.	Recovered fluid contained 2.8 per cent glucose, 1.6 per cent sucrose.
19A	3.2	Canned pineapple juice { 5 per cent glucose, 2 per cent sucrose	90	Cardia to ileocecal valve	30	180	210	Moribund	Fluid all in stomach. Intestines empty. Recovered fluid contained 1.4 per cent glucose, 1.7 per cent sucrose.
34	1.9	Mixed canned fruit juices (15 per cent glucose)	40	Cardia to ⅗ distance from ileocecal valve	15	55	70	Good	
34A	1.9	Mixed canned fruit juices (15 per cent glucose)	60	Cardia to ⅗ distance from ileocecal valve	10	116	120	Moribund	
35	3.3	Mixed canned fruit juices (15 per cent glucose)	40	Cardia to ⅗ distance from ileocecal valve	24	30	54	Fair	

the stomach and small intestine, although it would perhaps be more correct to correlate the volume in each instance with the weight of the animal. For a loop shorter than from the cardia to the ileocecal valve a somewhat smaller average volume might be taken as an adequate standard for distention, but for simplicity's sake this will be disregarded.

One may not, of course, ignore the distention produced by the accompanying fluid volume, which in numerous instances exceeds the gas volume, and for this reason the fluid volume as well as the total gas and fluid volume is included. The latter is, from the clinical point of view, perhaps the most significant of all, as it is the fullest measure of the general distending property of a food. However, in this study we are concerned with gaseous distention rather than total volumetric distention.

It should be noted that the same experiment may be repeated with the letter A, B or C attached. These refer in corresponding order to second, third or fourth experiments carried out in the same manner twenty-four hours after the preceding one, first emptying the loop of gas and fluid by stripping the contents into the stomach and then completely emptying the latter. Such additional experiments were carried out utilizing the same or some other food and either the same length of loop or a shorter one as indicated.

The condition of the animal before autopsy is included because in numerous instances it was an accurate reflection of the degree of harm inflicted by the particular food. In others it served as a crude basis upon which to judge the animal's capacity to deal with additional foods injected into an already badly damaged intestinal tract. This was, however, better determined in many instances by noting the extent to which the food had been digested or absorbed, reference to which is made among other things in the column labelled "Remarks."

Considering the data in the order presented, it is clear that glucose in 2 per cent and 5 per cent concentration in moderate quantity (40 c.c.)* caused no gas formation, or distention by fluid, and the animal remained in excellent condition. Presumably this can be attributed to the rapid absorption of these solutions. In experiments 1, 4 and 5 at least the entire ileum and some jejunum were excluded, so that this is some evidence that glucose in these concentrations can be absorbed in the upper small intestine.

Experiment 5A, in which 80 c.c. of 5 per cent glucose were given, did however produce distention. Possible explanations for this were: (1) this volume could not be speedily disposed of in a short high loop; (2) the efficiency of absorption by this loop was diminished because of (a) its age (twenty-four hours) or (b) the trauma incident to a second operation on the loop; or (3) to a combination of these factors. As Cannon and Murphy[8] demonstrated years ago, even slight handling of the gut delays peristaltic activity. If an animal is ill or shows a generally asthenic state, intestinal activity is likely to be correspondingly suppressed. Such an inhibition of intestinal activity must have prevailed to some degree at least in Experiment 5A in which the burden of a twenty-four-hour obstruction was augmented by a stripping of the gut before introducing the second dose of glucose. To this extent the glucose may have been slow in its exit from the stomach and its speed of absorption delayed. Such a situation must be even more pronounced in the case of foods of thicker consistency, which will not run so readily as liquids along the intestinal tract.

We would emphasize these points in advance of the presentation of further data relating to other foods studied under similar conditions, for it should be appreciated that the gas liberated by the interaction of a particular food with

* An average cat's stomach is moderately filled by 60 c.c. of fluid, well filled by 90 c.c. and distended by amounts exceeding 100 c.c.

TABLE XIII

GAS PRODUCTION IN CLOSED LOOPS BY FOODS

Exp. No.	Wt. of Cat (Kilos)	Type of Food	Amount of Food (C.c.)	Type of Loop	Gas in Loop (C.c)	Fluid in Loop (C.c.)	Total Gas and Fluid (C.c.)	Condition of Animal before Autopsy	Remarks
A. PROTEINS									
36	2.1	Gelatin (liquid)	70	Cardia to one foot from ileocecal valve	0	10	10	Excellent	Usual concentration of gelatin in water.
37	3.8	Gelatin (liquid)	90	Cardia to ileocecal valve	10	57	67	Good	3 teaspoonfuls gelatin to ¼ cup water.
37A	3.9	Gelatin (liquid)	90	Cardia to ileocecal valve	2	165	167	Died 48 hrs. after second op.	30 per cent by wt. of gelatin in water. Gas and liquid measured after death.
38	3.1	Albumen (raw egg white)	40	Cardia to ⅗ distance from ileocecal valve	2	39	41	Good	
39	2.9	Albumen (raw egg white)	60	Cardia to ileocecal valve	42	58	100	Good	
39A	2.9	Albumen (raw egg white)	70	Cardia to ⅕ distance from ileocecal valve	40	105	145	Good	
40	3.4	Meat (cooked lean hamburg)	60	Cardia to ileocecal valve	0	25	25	Good	Small incision in stomach necessary for introducing meat.
41	2.5	Meat (cooked lean hamburg)	60–90	Cardia to ileocecal valve	16	10	26	Good	Small incision in stomach necessary for introducing meat.
B. FATS									
42	3.5	Olive oil	30	Cardia to ⅗ distance from ileocecal valve	2	105	107	Good	Recovered fluid chiefly oil.
43	2.5	Chicken fat	60	Cardia to ⅗ distance from ileocecal valve	6	89	95	Good	Recovered fluid chiefly fat
44	2.5	Chicken fat	90	Cardia to ileocecal valve	3	128	131	Good	Recovered fluid chiefly fat.
45	2.3	Chicken fat	100	Cardia to ileocecal valve	2	140	142	Fair	Recovered fluid chiefly fat.
46	4.0	Cream (40 per cent)	100	Cardia to ileocecal valve	92	68	160	Good	Received fluid chiefly cream.
46A	4.0	Cream (40 per cent)	90	Cardia to one foot from ileocecal valve	44	155	199	Sick	Received fluid chiefly cream. Mild peritonitis.
47	3.5	Cream (40 per cent)	60	Cardia to ⅗ distance from ileocecal valve	40	70	110	Very sick	Recovered fluid chiefly cream. Mild peritonitis.
48	2.5	Cream (20 per cent)	60	Cardia to ⅕ distance from ileocecal valve	10	100	110	Very sick	Recovered fluid chiefly cream. Gangrenous ileal loop produced for other purposes.
C. MISCELLANEOUS FOODS									
49	?	Milk	40	Cardia to ileocecal valve	12	78	90	Moribund	
50	2.2	Milk	100	Cardia to ileocecal valve	320	185	505	Died 20 hrs. postop.	Autopsy immediately after death.
51	3.2	Milk	90	Cardia to ½ distance from ileocecal valve	328	108	436	Good	
52	2.3	Milk	40	Cardia to ½ distance from ileocecal valve	7	32	39	Seemed in shock	
53	2.6	Milk	40	Cardia to ⅖ distance from ileocecal valve	184	39	223	Found dead 16 hrs. postop.	
54	3.6	Milk	40	Cardia to ⅗ distance from ileocecal valve	91	19	110	Good	
23A	2.5	Milk	50	Cardia to ⅖ distance from ileocecal valve	109	74	183	Moribund	Mild peritonitis.
55	2.0	Butter milk	90	Cardia to ileocecal valve	68	0	68	Good	
55A	2.0	Butter milk	90	Cardia to ½ distance from ileocecal valve	22	80	102	Died 22 hrs. postop.	Gangrene of ileal loop produced for other purposes.

TABLE XIII (*Continued*)

Exp. No.	Wt. of Cat (Kilos)	Type of Food	Amount of Food (C.c.)	Type of Loop	Gas in Loop (C.c)	Fluid in Loop (C.c.)	Total Gas and Fluid (C.c.)	Condition of Animal before Autopsy	Remarks
C. MISCELLANEOUS FOODS (*Continued*)									
62B	2.0	Malted milk (50 per cent)	70	Cardia to ileocecal valve	8.	80.	88.	Very sick	Peritonitis in region of ileum. Third operation on this cat.
56	3.3	Malted milk (30 per cent)	90	Cardia to ileocecal valve	470.	40.	510.	Found dead 16 hrs. postop.	Death from distention. Autopsy hours later.
57	3.3	Malted milk (30 per cent)	90	Cardia to ileocecal valve	478.	130.	608.	Found dead 16 hrs. postop.	Death from distention. Autopsy hours later.
58	2.1	Egg yolk (raw)	75	Cardia to ileocecal valve	80.	85.	165.	Good	Very foul gas.
58A	2.1	Egg yolk (raw)	75	Cardia to ½ distance from ileocecal valve	134.	95.	229.	Died 20 hrs. postop.	Autopsy immediately. Recovered fluid mostly unchanged egg yolk. gas foul.
59	2.8	Whole egg (raw)	60	Cardia to ⅔ distance from ileocecal valve	50.	180.	230.	Good	
60	2.9	Whole egg (raw)	100	Cardia to ileocecal valve	65.	150.	215.	Very sick	
61	2.3	Custard (soft)	80	Cardia to 1 foot from ileocecal valve	530.	45.	575.	Died 20 hrs. postop.	Death from distention. Autopsy three hours later.
62	4.2	Custard (soft)	100	Cardia to ileocecal valve	210.	100.	310.	Very sick	Some unchanged custard in recovered fluid.
62A	4.2	Custard (soft)	90	Cardia to ileocecal valve	85.	72.	157.	Very sick	
63	4.1	Toast (puree)	80	Cardia to ileocecal valve	30.	20.	50.	Excellent	
64	3.0	Toast (puree)	80	Cardia to ileocecal valve	20.	3.	23.	Excellent	
64A	3.0	Toast (puree)	60	Cardia to 1 foot from ileocecal valve	0.	40.	40.	Very sick	Gangrenous ileal loop produced for other purposes.
65	2.3	Gruel (farina)	100	Cardia to ileocecal valve	45.	50.	95.	Good	
65A	2.3	Gruel (farina)	100	Cardia to ileocecal valve	30.	50.	80.	Sick	
66	3.5	Gruel (farina)	100	Cardia to ½ distance from ileocecal valve	28.	78.	106.	Good	
67	3.7	Macaroni	60	Cardia to ileocecal valve	30.	30.	60.	Good	
67A	3.7	Noodles	60	Cardia to 1 foot from ileocecal valve	26.	65.	91.	Died 20 hrs. postop.	Gangrenous ileal loop produced for other purposes.
68	4.0	Bean puree (lima)	40	Cardia to ileocecal valve	133.	80.	213.	Good	Autopsy 48 hrs. postop.
69	2.8	Bean puree (lima)	40	Cardia to ⅔ distance from ileocecal valve	155.	80.	235.	Good	
70	3.4	Bean puree (lima)	40	Cardia to ⅔ distance from ileocecal valve	241.	38.	282.	Good	
71	2.2	Bean puree (lima)	50	Cardia to ⅔ distance from ileocecal valve	14.	40.	54.	Good	Food unchanged, recovered from stomach.
72	?	Bean puree (lima)	60	Cardia to ⅔ distance from ileocecal valve	40.	55.	95.	Moribund	Food unchanged, recovered from stomach. Peritonitis.
73	2.2	Black coffee	90	Cardia to ileocecal valve	2.	2.	4.	Good	
74	3.6	Mineral oil	40	Cardia to ileocecal valve	2.	70.	72.	Good	
75	4.6	Magnesium sulphate (25 per cent)	20	Cardia to ½ distance from ileocecal valve	5.	165.	170.	Good	
76	4.2	Magnesium sulphate (25 per cent)	40	Cardia to ⅔ distance from ileocecal valve	1.	185.	186.	Very sick	
77	?	Magnesium sulphate (25 per cent)	40	Cardia to ⅔ distance from ileocecal valve	4.	170.	174.	Very sick	

intestinal contents in its progress along the gut will vary according to (1) the general condition of the animal; (2) the degree of suppression of intestinal secretions; (3) the amount of fluid available from the blood for diluting hypertonic solutions, and (4) the variable degree of damage to the gut with a correspondingly varying amount of inhibition of intestinal activity and capacity for recovery within the period of experimental observation. It will therefore not be surprising if uniform results are not always obtained with the same food, although a tendency in one direction or another will be shown to exist.

It can be safely concluded that glucose in 5 per cent (or lower) concentration is not likely to prove embarrassing to the intestine, whether by gas or fluid, even when the ileus is located at a relatively high level, unless the volume administered is excessive, or the function of the gut badly compromised.

When solutions of 10 per cent glucose were tested the result was gaseous distention in 3 out of 6 instances, although, to be sure, relatively large amounts were necessary to produce it. It is worth pointing out that Cat 7 was able to dispose of 100 c.c. rather comfortably, but when on the following day the same amount was given, and only half as much absorbing surface made available, death resulted from distention. In Cat 9 the first dose produced considerable gas, but doubling the dose with the same absorbing surface produced on the second day gross distention. Whether the decreased absorbing surface in Cat 7, or the doubled dose in Cat 9, or the severely traumatized gut in both, caused the distention is not so material as that these factors may be singly or in combination decisive factors. Cat 6, however, will testify that the amount of glucose alone may be enough to cause great damage.

This is more emphatically demonstrated by 50 per cent glucose. Forty c.c. caused huge distention in 4 animals, 2 of which died in less than sixteen hours. In a fifth, a little more than average distention occurred, and in 2 more with only 20 c.c., a little less than average. All these animals were in excellent condition before operation. From the comments under the heading "Condition of Animal before Autopsy" the effect of such solutions compared with weaker ones is plain.

In Cat 26 c there was an atypical response, the explanation for which is simple. The cat had had an operation on each of three preceding days with stripping of the gut each time; there was peritonitis; and the glucose remained, except for some dilution, unmodified in the stomach. This cat illustrates a point previously referred to and repeatedly observed with other foods which ordinarily were capable of causing distention, namely, that in a very sick animal there may simply be no reaction. The food lies, as it were, in an indifferent container.

In different animals with the same amount of 50 per cent glucose one does not find a quantitative correlation between the extent of gas formation and the amount of absorbing surface available. Even when the ileum is accessible gas may form in large amounts.

No close correlation between volumes of gas and fluid appeared in these experiments, nor in others with different foods. Reasons for this are such factors as the degree of muscle tone and peristaltic activity, and the capacity for dilution and absorption which the particular animal happens to be capable of at the time of the experiment.

Only a few 50 per cent cane sugar determinations were made. We expected a response similar to that of 50 per cent glucose, but obtained it in only 2 out of 5 instances. In one animal, though the largest in the whole series of experiments, the quantitative result, 750 c.c. of gas and fluid, was truly remarkable. It is notable that this cat was in good condition when the determination was made. In those which did not form gas the explanation

may lie in the fact that whereas nearly all strains of the colon-typhoid group of bacteria ferment glucose, only B. coli communior and B. lactis aerogenes ferment sucrose.

Five animals with grape-juice, which contained 17 per cent dextrose and 1 per cent cane sugar, gave 2 instances of enormous distention, while in 3 others none occurred. It will be noted that in the latter three the ileum was included in the loop, but in view of the results with the glucose experiments it cannot be confidently stated that the ileum, by virtue of its greater absorbing capacity, acted, so to speak, as a safety valve.

Orange juice demonstrated a rather striking capacity for producing meteorism. One notes again that increasing the dose in the same animal (Exp. 20, 20 A; 23, 23 A), or using a large dose (100 c.c. Exp. 21) to begin with, is more likely to produce distention, although the variable reaction of two animals to the same dose is well illustrated by comparing Experiments 24 and 25. The explanation for gaseous distention by orange juice is somewhat obscure in view of low dextrose (3.4 per cent) and sucrose (1.75 per cent) content, unless the cellulose (the orange juice was strained) can be a source of consequence.

A sample of ginger ale, containing 1 per cent glucose and 3.8 per cent sucrose, was administered twice to each of 3 cats. Gross distention occurred on both occasions in one, only on the second attempt in another, while in the third, a much larger cat, none resulted with the first dose (90 c.c.) or with the second one which was almost twice as great. In this cat the ginger ale had been decarbonated, while in the first two it had not, so that it is possible that distention, due to diffusion of N_2 from the blood (see text below), may have occurred in the first two because absorption of CO_2 could not keep pace with its rate of liberation.

A variety of canned fruit juices (about 5 per cent glucose) in fresh and compromised loops caused no distention except in one case. In this experiment (93 A), as well as in several others with different foods (Exp. 32 A, 23 A, 15) the gas and fluid were not measured until several hours after death, so that some of the gas may have formed post mortem, although distention seems to have been the cause of death.

To summarize, it is accordingly to be concluded: (1) that gaseous distention of striking proportions can be caused by a variety of carbohydrate solutions in common use in the liquid diet stage of the postoperative period; (2) that the degree of distention is not necessarily in proportion to the concentration of glucose, though the trend seems to be in that direction; (3) that whether or not distention will occur cannot be safely predicted, since it will depend on factors variable in each individual. It is probable that the more nearly normal the gastrointestinal tract the better will it be able to dispose of these foods and avoid distention due to them. On the other hand, a very sick animal with practically total suppression of intestinal function will likewise not distend because of complete failure to react to the presence of food, especially if the food fails to be expelled from the stomach into the small intestine.

The volume of solution used in these cats to produce distention is not out of proportion to the average volume per kilogram consumed in twenty-four hours by the average postoperative patient.

Since the data were obtained by producing a mechanical ileus, the figures are a more accurate reflection of what may happen in such a case than in the functional ileus of the immediate postoperative period, where the stasis and resulting delayed absorption of fluids with consequently greater gas formation is presumably not nearly so marked.* Nevertheless we feel it correct to assume that the tendency to gas formation by foods as noted in these

* Alvarez[6] has pointed out that fluids will run fairly easily in any direction along the gut so long as there is no complete mechanical block.

experiments is an adequate approximation of their behavior in any type of ileus.

2. *Proteins* (*Table* XIII, *Section* A). Three varieties of pure proteins were tried in 6 different animals with uniformly negative results. It is interesting to note in the column "Condition of Animal before Autopsy" the relatively innocuous effect of these foods, although Cat 37A died 48 hours after the second experiment. One objection to the validity of these observations may lie in the supposition that whereas the simpler carbohydrates require little enzymatic activity for their disposal, proteins have not time within the duration of these experiments to react with intestinal juices, owing to the probable suppression of secretory activity induced by the operation. Cannon and Murphy showed that this suppression of activity may indeed last for six or more hours postoperative, but it is hardly likely that this would persist for 24 hours. In any case, except for repeat experiments 37A and 39A, the residual fluid removed was of a character to indicate that reaction had indeed taken place and absorption to a large degree effected.

3. *Fats* (*Table* XIII, *Section* B). The action of fats is analogous to that of proteins. Only in one of eight experiments (No. 46, 40 per cent cream) did distention appear. Contrary to the proteins and carbohydrates, the fats used were all recovered practically unchanged except for dilution. It has long been known that these substances slow up peristalsis, but the absence of gas cannot be ascribed to their failure to reach the small intestine since they were found filling the loop down to the level of the ligature, as was true in nearly all other experiments in which the animal was in reasonably good condition.

The absence of bile was notable in these experiments, although it was commonly found in the recovered fluids of other experiments. This may be the reason for failure of the fats to become absorbed. The prolonged residence of fats in the intestine may act as an inhibiting influence on the digestion of carbohydrates simultaneously ingested, and thus serve partly as an explanation for the frequent intolerance for sweets displayed by victims of biliary tract disease. Similarly, the "bloating" complained of by many dyspeptics after eating candy or rich desserts might be accounted for by a slowed absorption with resulting increased opportunity for fermentation and gas formation.

4. *Miscellaneous Foods* (*Table* XIII, *Section* C). Milk proved to be a notorious offender. Five of 7 cats were victims of distention, the magnitude of which paralleled the volume used. It is noteworthy that only 2 cats of the series seemed in good condition the day following operation, 2 others died and 3 others were moribund when electrocuted. We had a distinct impression that milk was the most treacherous of all the foods used, not alone because of distention, but because of some other unaccountable lethal effect.

Buttermilk tried twice on the same animal caused no distention, but malted milk killed 2 healthy animals from gaseous distention in less than sixteen hours, while a third did not react at all because it was the third successive experiment in the same cat with peritonitis setting in before the malted milk was injected. Custard, which is chiefly milk with some egg and sugar, in one cat generated 530 c.c. of gas. In a second cat 210 c.c. formed in the first test, which made the animal very sick. Repeating it in this animal caused distention somewhat below the average standard. We attributed the effect of the custard to its milk content, because (1) the amount of sugar was small, and (2) whole egg in 2 cats failed to cause average distention although egg yolk tried twice in one cat did produce average distention.

In view of these results it is clear that milk in pure or mixed form may be a serious source of danger in any case of disordered intestinal function. Its widespread use with impunity in cases of gastric surgery may be on account of the relatively late stage after operation before

it, or any other food, is allowed.

In eight experiments carbohydrate food in the form of toast, gruel, macaroni or noodles, displayed a quite benign behavior. The gas volume was small and the animal's condition remained good except in three repeat experiments (58A, 59A, and 61A) in which the sick state of the cat was due to the duration of the obstruction.

The remaining experiments may be summarized in a few words. Puree of lima bean, a leguminous food, was found to be a vigorous gas producer on three occasions. In two, when it failed to do so, the food was found unchanged in the stomach. One experiment with black coffee was completely negative. The complete disappearance of the injected fluid suggested, as did the 5 per cent glucose experiments, that the loop as used in our experiments was capable of absorption until the deleterious effects of obstruction became operative. Mineral oil gave a negative result for gas in one animal. Three trials with 25 per cent magnesium sulphate solution were likewise negative for gas, but the fluid distention resulting was considerable. These observations served as a check on other experiments with fluids of high osmotic tension (e.g. 50 per cent glucose), showing that stretching the gut wall by fluids is not adequate to cause an accumulation of gas.

An analysis was done of the gases generated by several of the substances listed in Tables XII and XIII.* It will be recalled that the only absorbable gases for consideration in distention are O_2 and CO_2 (H_2S, if generated, being quantitatively absorbed). In a preliminary analysis of a gas sample in a 50 per cent glucose experiment (Exp. 11, Table XII), the percentage of CO_2 was 14.6 per cent, of O_2 1.4 per cent, leaving a relatively non-absorbable residue of 84.0 per cent. Another sample from a stomach loop containing 50 per cent glucose (Exp. 4, Table X) yielded 12.4 per cent CO_2, 3.9 per cent O_2, and a relatively non-absorbable residue of 83.7 per cent. Further analyses gave the results tabulated below (Table XIV).

TABLE XIV

PERCENTAGE ANALYSIS OF GAS SAMPLES PRODUCED BY FOODS

Experiment Number (From Tables XII and XIII)	*Type of Food*	*Total Gas Volume (C.c.)*	CO_2	O_2	N_2	H_2.....CH_4 *Combustible Residue**	N_2, H_2 & CH_4 *(Total Non-absorbable Fraction)*	*Condition of Anima when Gas Was Recovered*
28	Orange juice	42	12.7	?	53.0	34.2	87.2	Fair
60	Whole egg	65	21.6	0.14	68.9	9.3	78.2	Poor
9A	10 per cent glucose	90	16.2	2.8	70.6	9.8 — 0	80.4	Good
54	Milk	91	11.6	?	66.4	22.0	88.4	Good
29	Orange juice	148	9.8	?	27.0	63.2	90.2	Fair
69	Bean puree	155	9.2	0.27	30.1	60.2	90.3	Good
70	Bean puree	244	6.6	?	33.5	59.9	93.4	Good
23A	Milk	109	17.9	0.20	4.6	68.9 — 8.4	81.9	Moribund
50	Milk	320	13.2	0	52.5	34.3 — 0	86.8	Died; gases removed immediately
15	50 per cent glucose	291	39.4	?	6.8	55.4	62.2	Dead some hours
57	Malted milk	478	36.6	0	0.4	62.9	63.3	Dead some hours
61	Custard	530	29.3	0.34	5.9	63.5 — 0	69.4	Dead three hours

* The figures in the middle column are for percentage totals of both gases when individual analyses were not made.

It will be noticed that in those cats from which the gas sample for analysis was removed while the condition of the animal was fair or good, the percentage CO_2 varied from 6.6 to 16.2 per cent. In three which had been dead some hours it varied from 29.3 to 39.4 per cent, obviously because of continued gas formation post mortem. Consequently it appears that this gas, which is formed in vastly greater quantity than any other, will not be a

* We wish to acknowledge our gratitude to Dr. George Robb of the Thorndike Memorial Laboratory at the Boston City Hospital for his very kind advice and help in carrying out some of the gas analyses.

major constituent of the gases due to foods. This is in accord with all previous studies which demonstrate the very high diffusibility of this gas. But with pathological states arising to interfere with its absorption, as in mesenteric thrombosis or in strangulation, it might form a significant portion of the distending gases developing from the breakdown of foods. However, the distention observable in cardiac decompensation, pneumonia, etc. (as Schoen,[2] McIver[1] and others point out) cannot be due to CO_2 since there is no reason to suppose that the extreme degree of venous block required to prevent resorption of CO_2 from the small intestine into the blood exists in these conditions. Once distention from any cause has become sufficiently advanced to fully compress the veins in the intestinal wall, CO_2 may become an embarrassing additional burden. This situation is simulated in the case of those animals which had died before the gas sample was removed (Exp. 57, 15, 61). They showed an abnormally high percentage of CO_2 because gas formation could proceed without simultaneous resorption.

Unfortunately we did not determine the percentage of O_2 in all cases. Nevertheless, in the preliminary analyses as well as in a few listed in Table XIV, this gas never exceeded 3.9 per cent. While somewhat higher percentages of this gas did diffuse out of the blood in some of the loop studies of McIver, Redfield and Benedict,[3] it can be said of O_2 that it is a negligible participant in gaseous distention.

Since H_2S in this connection can also be dismissed, there remain H_2, CH_4 and N_2 for consideration. Although the relative rates of absorption of these gases can be roughly expressed in a 7:4:1 ratio,[3] they may all be considered for practical purposes non-absorbable gases which for the most part are disposed of by expulsion. If, for convenience, they are grouped together as the nonabsorbable fraction of a given gas volume, it will be noted from Table XIV that they constitute 78 per cent or more of the total in all 8 animals from which the gas samples were taken antemortem, thus establishing the fact that the bulk of the distention generated by foods in our experiments was primarily due to gases which would in a case of ileus require removal largely by the restoration of normal peristaltic function.

In the differential analysis of this fraction N_2 varied from 27.0 to 70.6 per cent, if we exclude the figures for the dead or moribund animals.

There are only two possible sources of N_2 in intestinal gases: (a) from swallowed air, which was excluded in these experiments, and (b), as McIver, Redfield and Benedict[3] have already demonstrated, from the blood by diffusion. What conditions within the intestinal lumen are conducive to the diffusion of N_2 into it? In our experiments with closed empty loops, gas of sufficient volume to be significant for distention never collected. The experiments with magnesium sulphate in closed loops further demonstrated that the mere presence of a large fluid volume in the gut is not adequate cause for the collection of any gas, although, to be sure, some diffused gas may have been in physical solution in the fluid. But, as in McIver's experiments,[3] when the lumen of the gut is mildly distended with other gases, N_2 (and O_2 and CO_2) will diffuse into the loop in accordance with the difference in the partial pressure of this gas between the blood and the intestinal lumen. Hence the postulate is inescapable that N_2 will diffuse into the intestinal lumen from the blood as a free gas only as the result of the accumulation of other gases. The actual volume of N_2 entering the intestine will depend not only on its partial pressure in the blood, but also on the gas capacity of the intestinal lumen established by other gases generated within the intestine.*

* In 1897 Evans,[9] in attempting an explanation of the rapid production of distention in peritonitis, gastritis, hysteria and certain cardiac and pulmonary disorders, argued that N_2 in the gut is not necessarily of oral origin but is probably diffused out of the blood. As evidence he cited the observation that the "sound" or swim bladder of the fish, which has no connection

Hence the greater the gaseous distention of the gut the more N_2 will enter until equilibrium with its partial pressure in the blood ($\frac{4}{5}$ of an atmosphere) is reached.

So long as the intraintestinal pressure does not exceed one atmosphere, the volume of N_2 will accordingly tend to become 80 per cent of the total gas volume. Only the continued formation of other gases prevents this equilibrium from being reached, and the more rapidly these other gases are generated the further away from equilibrium will the percentage of N_2 be. If the rate of formation of other gases is slow, which is likely to be the case when the total gas volume is low, the percentage of N_2 will be closer to 80 per cent. This will be obvious from a comparison of the total gas volume with the percentage of N_2 in the first seven experiments of Table XIV.

Nitrogen must therefore be considered as one of the chief components of the distending gases. The only conceivable obstacle to its diffusion out of the blood in significant volume would be a rate of generation of other distending gases, e.g. H_2, such as to rapidly obliterate the capillaries in the gut wall by the excessive intraluminary tension. It is in this way that the very low actual (19, 1 and 39 c.c.) and relative volumes (6.8, 0.4 and 5.9 per cent) of N_2 found in cats 15, 57 and 61, which died very quickly after operation, can be accounted for.

In view of the paramount importance of N_2 as a distending agent, whatever its origin, an attempt to facilitate its reabsorption from the intestine into the blood might be considered. That this is feasible is suggested by several observations. McIver[3] found that a cat breathing pure O_2 resorbs pure N_2 from an intestinal loop much more efficiently than when breathing air, because N_2 is thereby washed out of the blood through the lungs. Alvarez[6] refers to Boothby's observation that pneumonia patients, while in an oxygen chamber, are not troubled with intestinal distention. Efforts to apply this treatment to other types of obstinate distention urgently needing relief would seem worth a trial.

There remains to point out in connection with our observations on the gas analyses that, of all the gases, H_2 is the chief offending agent in gaseous distention, since N_2 collects only secondary to the accumulation of other non-absorbable gases, and since the only other gas in this category is CH_4, which has been found to comprise a low or negligible fraction of the total non-absorbable fraction (see Table XIV). Boycott and Damant[14] found that in goats on a diet of hay and oats the combustible gases consisted of two-thirds CH_4 and one-third H_2, the former arising from the fermentation of cellulose and the latter from other carbohydrates. They stated that the CH_4 might be split further to form H_2 and CO_2. We did not study cellulose-containing foods except bean puree. Unfortunately in the one gas sample from this food we did not determine the methane.

SUMMARY AND CONCLUSIONS

In the starving cat gaseous distention of the stomach and small intestine will not occur as the result of a simple laparotomy. This is the case regardless of the degree of

with the intestinal tract, contains 80 per cent or so of N_2. He believed that an active secretion of N_2 from the blood occurred. Crookshank[10] also favored this idea, and more recently Kantor and Marks[11] accounted for the meteorism of pneumonia, spinal cord injuries, typhoid, etc., on a similar basis, postulating as a necessary adjunct to the secretion theory a condition of intestinal atony. On the basis of determinations of the rate of CO_2 resorption in closed loops under pilocarpine and atropine, Schoen[2] concluded that within certain limits the efficiency of gas absorption increases with the tone of the intestinal muscle. The converse of this, namely, that the lower the tone of the muscle the less gas will be absorbed, is invoked by Schoen, as well as by Kantor and Marks, as the explanation in part of the meteorism in shock, pneumonia, peritonitis, and other severe infections. As the distention increases atony ensues and still further depresses the resorption rate.

It does not seem reasonable that atony can be considered a significant fault since Hotz[12] and Olivecrona[13] have demonstrated that even in severe peritonitis muscle tone is well preserved and that contractions cease only after prolonged distention by gas. The loss of tone is conditional upon an overstretching by gas and is therefore merely an end result of an extreme state.

trauma inflicted on the gastrointestinal tract, providing it is left structurally intact.

When mechanical obstruction is produced in a starving cat, gaseous distention may or may not result, depending only on the amount of air the animal swallowed. Gases of local origin do not collect in these animals in more than negligible volume and are, therefore, not a factor in the resulting distention. The collection of fluid in these animals is dependent primarily on the accumulation of gas, though not necessarily in large volume. Although these observations are not directly applicable to a state of postoperative functional ileus, we feel that they still reflect the conditions causing distention in the latter. It is, therefore, concluded that in the absence of food, swallowed air is the only significant source of gaseous distention of the intestine. This is true even when the circulation of the intestine is severely injured.

In addition to swallowed air the type of food present in the gastrointestinal tract after operation must be considered as a possible source of gaseous distention. Our experiments show that liquid carbohydrates are a particularly important source of distending gases. The higher the glucose content of these carbohydrates the more gas is likely to form, although certain ones containing not more than 5 per cent glucose may also produce severe grades of distention. Of the latter, orange juice and ginger ale deserve special mention. Other foods capable of causing significant degrees of gaseous distention are milk, whether pure or in mixed form, and foods rich in cellulose, such as bean puree. Pure proteins, fats, and certain carbohydrates such as toast, gruel, macaroni, etc. are not sources of significant gas formation.

Distention does not occur in every instance in which gas-producing foods are present on account of a number of variable factors, such as the general condition of the animal, the amount of food, and the state of motor, secretory and absorptive activity in the intestine. Consequently it should not be surprising to find widely varying degrees of distention with the same type of operation and similar preoperative and postoperative management.

Analysis of the gases produced by foods showed: (1) that nitrogen and hydrogen constitute 80 per cent or more of the total gas volume; (2) that nitrogen diffuses out of the blood stream in such volume as to approach an 80 per cent fraction of the total gas content, but the extent to which it succeeds varies inversely with the rate of accumulation of other gases; and (3) that since the diffusion of nitrogen into the gut will not occur in significant volume unless other gases have first accumulated, the distention resulting from foods must be caused primarily by relatively non-absorbable gases of which hydrogen is the most important volumetrically.

Our observations suggest that in the treatment of distention, the baneful influence of certain foods must be considered along with the probably equally vital role of swallowed air. The necessity of administering liquids and carbohydrates in the immediate postoperative period can be met by the parenteral administration of glucose solutions, while the desirability of reestablishing normal peristalsis, which is better initiated by the resort to solid or semisolid foods, would be more safely and effectively accomplished by the use of such foods as toast, gruel or other cooked cereals, egg albumen and other non-gas producers within a reasonable time after nausea and vomiting have subsided.

The technical assistance of Mr. Samuel Levinson is gratefully acknowledged.

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[For Remainder of References see p. 213.]

ANATOMICO-PHYSIOLOGICAL CONSIDERATIONS IN THE TREATMENT OF TUBERCULOSIS OF THE SPINE

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THE marked curative effect of bony ankylosis upon any tuberculous joint is axiomatic in orthopedic surgery. Without ankylosis, an advanced tuberculous process in a joint can never be said to be cured. In planning treatment of tuberculous spines, it is therefore of extreme significance that, anatomically, we are dealing with joints under constant uncontrollable mechanical influences, such as respiration and involuntary muscle spasm. This fact contributes greatly to the spread of the tuberculous process, and operates against cure. It not only makes it more difficult for Nature to set up a protective ankylotic process, but also presents increasing difficulties to the surgeon who attempts to bring about immobilization by non-operative conservative measures.

These non-operative measures include the application of casts and braces and the use of the frame and the block. The plaster cast must be judged according to the general principles of splinting. Since both thorax and abdomen normally change their dimensions constantly, the cast cannot be fitted closely enough to maintain immobility, and this cardinal principle of splinting must be sacrificed. It cannot be expected that any form of external splinting will immobilize a series of short bones of irregular contour to which are attached muscles acting in co-ordination with the muscles of respiration and the voluntary muscles which move the head and limbs. An added disadvantage of these imperfect attempts at immobilization is that the cast which is applied to accomplish cure seriously restricts the movements of the thorax and abdomen and interferes with respiration, oxidation and nutrition on which so much depends in the treatment of any form of tuberculosis. This is in itself paradoxical.

If the cast is applied to a child during the period of growth, the tendency to produce muscle and bone atrophy by prolonged recumbency and inactivity is most unfortunate biologically, anatomically and physiologically, and, in my opinion, far outweighs any objection to submitting a child to an operation of so slight magnitude with such outstanding curative influence, as opposed to prolonged conservative treatment which so often is followed in a year or two by relapse. If the case is referred for operation at this stage, the disease has frequently progressed to such a degree that deformity cannot be entirely prevented. The late end results of cases in which the bone-graft operation for Pott's disease was performed in early childhood are so favorable, and I have seen so many tragic cases of unsuccessful results from prolonged conservative treatment, where operation has been sought too late, that I stand emphatically for early operation. Recent literature shows that more and more surgeons are inclining toward this view.

If there is an apical focus, the cast tends to aggravate it by constricting the lower lobes and leaving the upper relatively free, thus throwing increased mobility and work on the diseased portions of the lungs. The extent to which distortion of the abdominal contents may be carried is illustrated by the extreme degree in which the abdomen sometimes rolls out from beneath the cast.

The Bradford frame and the block are not open to all these objections but they have their own disadvantages, the most important of which is the protracted

inactivity of the patient, and the fact that one never can be certain there will not be relapse so long as there is no bony ankylosis.

One therefore turns to internal immobilization by means of an autogenous bone graft as the procedure of choice. My first attempts, in 1909, were directed toward fusion of the spinous processes without using bone-graft material from elsewhere. I was not satisfied with the ultimate results, and the more I examined the principles on which the operation was based, the more faulty did this procedure seem. The spinous processes supplied an insufficient amount of bone to anchor the vertebrae securely. Of greater fundamental importance was the consideration of the extremely low osteogenic potentiality of vertebral bone.

We know clinically that vertebral bone has a low degree of reparative potentiality as evidenced by the following phenomena. The progressive lysis of traumatized vertebrae as evidenced in Kummel's disease suggests that the bodies have little recuperative osteogenic power, and the progression of tuberculosis in the vertebral body is apparently not hindered by any adequate reparative process of osteogenic nature. Secondly, when the lateral process is broken, although the fragments may lie in perfect apposition, they do not unite in more than 5 per cent of cases. Spies, who has made a special study of fracture of the articular processes, states that they rarely unite when fractured. Furthermore, marked lack of reparative reaction is shown when the vertebral bodies are involved by tuberculosis. In many instances I have seen as many as eight or ten vertebral bodies entirely disappear without any apparent effort at bone replacement. The *x*-rays have shown not only the complete disappearance of these bodies, but no bone growth or callus whatsoever.

These clinical observations may be explained by the fact that, embryologically, vertebral bone is derived from the ectoderm from which tissues of higher differentiation have their origin, and all of them have a low degree of reparative ability. One need only mention the optic nerve, the nerves of hearing and those of the spinal cord as illustrations. This may be Nature's plan that vertebral bone should have a low degree of osteogenesis. For although this may operate to great surgical disadvantage, yet for Nature's purposes it is much safer than if osteogenesis were high; because when spinal bone is involved by disease or fracture, it does not pour out callus which might encroach upon the spinal cord which the spinal bone is protecting.

These convictions regarding the low osteogenetic potentialities of vertebral bone were confirmed by my early animal research experiments in bone growth in 1908 and 1909.[1] Cubes of vertebral bone and ulnar bone taken from the same dog were transplanted into his muscle under identical conditions, and after a period of weeks the ulnar bone showed marked superiority in osteogenetic power, both microscopically and macroscopically, as compared with the vertebral bone.

Because of this inadequate callus-forming capability of vertebral bone it is clear that, in order to secure immediate strong fusion, it is more trustworthy to insert a strong single graft from a long bone of high osteogenesis to supplement that of the local vertebral bone. Furthermore, a massive bone-graft splint provides immediate immobilization of the affected vertebrae. When I first used the spinal graft, I chose autogenous bone from the patient's tibia for reasons which seemed then, and I am convinced still are, in accord with the soundest biologic principles.

The tibia, fortunately for the purpose of the reconstruction surgeon, inherently has great osteogenetic potentiality and excellent reparative power. When inlaid into the spinous processes, it not only serves as an excellent internal immobilization splint but immediately fuses, by its own osteogenetic influence and that of the spi-

nous processes themselves with the spinal column. A second very important curative consideration is the aid to vascularization

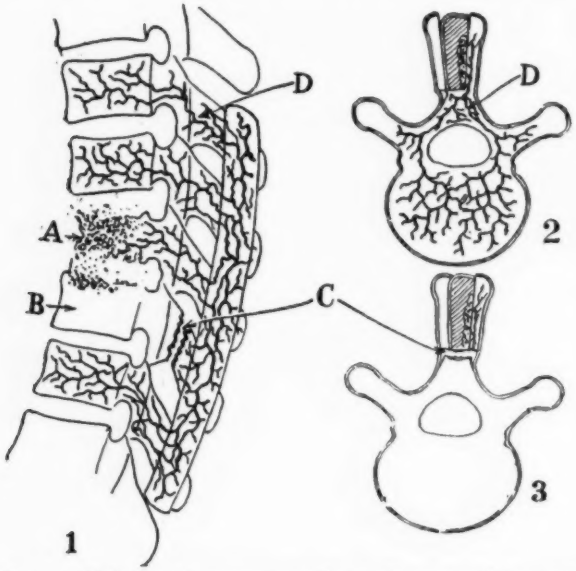

Fig. 1. Schematic representation of supporting and vascularizing functions of graft. Section shows side of gutter on which half-spines are left intact (D). Circulation in graft has linked up with that of healthy vertebrae and with one diseased vertebra (A) through intact half-spine; diseased vertebra (B) fails to obtain immediate mechanical support or revascularization because half-spine has been fractured (at C) and has not reunited. This is illustrated to show the disadvantage of fracturing the second half either by accident or by design.

of the tuberculosis-involved portion of the vertebrae by means of the vascular canalization of the massive bone graft.

My hope was that this graft of living bone would not only provide immediate mechanical support and added osteogenetic power to promote ankylosis but would also act as a vascular-conducting scaffold from each spinous process to the next, and from the unaffected portion of the vertebra to the portion whose circulation is impaired by the effect of the tuberculosis. A diagrammatic representation of this is shown in Figure 1.

The blood supply to the cross section of long bones is 25 per cent through the periosteum and 75 per cent via the marrow and endosteum. In the case of flat or irregularly shaped bones such as the vertebrae, this relationship may be somewhat changed, but it is roughly in this proportion; and therefore the ideal procedure would be to contact the graft generously with the interior of the bone of the spinous process, the whole central portion of which is cancellous. This should be brought about with the minimum disturbance of the circulation to the bone into which the graft is being incorporated or inlaid. It was with this consideration in mind that my autogenous inlay graft for the treatment of Pott's disease, as well as other conditions of the spine, was designed. The spinous processes as well as their imbedding ligaments are split as near as possible to the median raphe with a scalpel and osteotome and into this cleft or gutter the massive graft is inserted.

In twenty years' use of the bone graft in tuberculous spines of all degrees of severity, both as to acuteness and extent of the disease, I have been increasingly impressed with the unusually favorable reaction and its curative effect when properly inserted. When I first designed the bone-graft operation for the spine, I believed, as did others, that it derived its beneficial influence solely from its immobilizing effect. During these years in studying a rapidly increasing series of cases by *x*-ray and an occasional postmorten case, I have been much interested in the unusual reparative and curative influence exerted by the ankylosis secured by bone graft; and my firm conviction has grown that there is something underlying this influence other than just mechanical immobilization.

I believe the amalgamation of diseased vertebrae with healthy ones on either side influences the repair of the diseased vertebrae at least in two ways; namely, increased stress with its stimulus to metabolism is thus brought to bear upon the undiseased portion of the involved vertebrae with complete immobilization of one vertebrae to another, and the bony bridge produced by the graft acts as a vascularizing influence, bringing blood from the posterior part of the uninvolved vertebra to the posterior part of the dis-

eased vertebral body, and thus to the diseased bone. This autogenous graft is put into the split spinous process in intimate contact with the vascular system of the vertebrae under the most favorable conditions for vascularization of the graft and its linking up with the vascular system of the vertebra. The immediate and profuse vascularization of a graft when inserted for any condition whatsoever has been a most striking phenomenon.

When I was in England in 1929, Sir Arthur Keith, the great clinical pathologist, wrote me that he had some specimens at the Museum of the Royal College of Surgeons which he would like to show me. Upon complying with his request, I was much impressed with the pronounced vascularization of specimens of grafts inserted for various purposes which he had obtained from post mortems and injected and prepared with the particular purpose in mind of demonstrating this phenomenon.

The early and complete vascularization of a graft has been very striking and interesting to me since my early animal experimental work in 1910, and I believe it to be one of the underlying causes of the extremely beneficial action of the bone graft when inlaid in the spine or through other tuberculous joints such as the knee, the tarsus, etc. This factor emphasizes the importance of accuracy of fit of the graft and host tissues, and also the importance of using a massive graft of ample length, as such a degree of beneficial vascularization could not be accomplished by bone chips. Under such conditions the Haversian canals of the graft very rapidly become canalized and increased in size in precisely the same way as we get a collateral circulation established when important blood vessels have been cut or occluded.

The osteogenetic potentiality of vertebral bone is always diminished by tuberculous involvement. Furthermore, the osteogenetic potentialities of different individuals vary within wide limits and are absolutely impossible to determine, even at the operating table. For these combined reasons, it is incumbent upon the surgeon to play the game as safely as possible in relation to a large series of cases; and to select not only an ample amount of bone in the form of a complete splint but bone of the greatest known osteogenetic potentiality. To rely on the low osteogenetic potentialities of vertebral bone, particularly in the form of chips, is to gamble on getting a favorable outcome instead of being practically certain of it.

The autopsy findings of Professor T. Wingate Todd, Professor of Anatomy at Western Reserve University, in a case of Pott's disease, further demonstrates the osteogenetic potentialities of the tibial graft as used by the author.

> The case is one of perfect functional result from an Albee operation. . . . Where the splint is grafted into the spinous processes, there has been marginal erosion and proliferation of new bone precisely as in the repair of a fracture. Where the splint lies imbedded in soft tissues, no proliferation or change has taken place in the implanted bone, except that a modification of its texture has occurred indicating a living but inactive condition. Hence both current views are justified regarding the fate of the splint, namely, that it remains unchanged and also that it acts as a temporary scaffolding for the erection upon it of new bone tissue.

Anatomically and mechanically we have vertebrae moving through an anterior-posterior excursion over the articular processes as an axis as the spine flexes and extends. These processes being separated a considerable distance apart, a broad fulcrum is thus afforded at the lateral facets. The graft is put in posterior to this fulcrum and serves to hold together the spinous processes or the posterior arms of the vertebrae considered as levers. This relieves all eroding pressure from the vertebral bodies which are, mechanically speaking, the anterior lever arms. It is a fortunate circumstance that the bone involved in this broad fulcrum as well as the spinous process or leverage bone in which the graft is inserted is practically

never affected by the tuberculous process.

An important consideration is the length of time required for an operation and the shock occasioned by it. An operation that consumes little time and induces comparatively little shock may obviously be undertaken when the surgical risk is not of the best. The bone-graft operation may be completed in as little as nine minutes in a favorable case, if one's operating team is thoroughly familiar with the routine and the special instruments required. Cannon and Turck have attributed shock to biochemical rather than to neurogenic or hematogenic factors. From macerated living tissue they have extracted a principle, the injection of which into the circulation, even in minute quantities, precipitates profound shock. Laceration of tissue by trauma or at operation thus releases the poison that, when entering the circulation, produces a lowering of blood pressure and shock. Rough handling and violent retraction at operation add to the effect by forcing this poison into the circulation. The technique does not require excessive retraction and there is no necessity to operate more or less blindly in regions difficult of access. Hence the speed of the operation and the comparative freedom from shock. The small amount of shock produced by this operation is due to its simplicity and consequent shortness of duration as well as the minimum amount of retraction and traumatization of tissue it entails.

INDICATIONS AND CONTRAINDICATIONS FOR THE OPERATION

My practice is to operate on any patient at any age provided the surgical risk is good. The surgical risk in this instance depends on several factors:

(1) *General Condition of the Patient.* I have already discussed the question of shock in relation to different operations for tuberculosis of the spine. If the surgeon is skilful and rapid, and works in suitable surroundings, he may consider the bone-graft operation safe if the patient is fit for any major operation.

Paraplegia and perivertebral and psoas abscesses provide an additional indication for the operation.

High temperature due to secondary pyogenic infection is a contraindication to operation. The bacteriemia must be allowed sufficient time to subside; a transfusion of blood may help. Pyrexia due solely to the tuberculosis is not a contraindication to operation.

(2) *Multiple Lesions.* The presence of tuberculous foci elsewhere has often to be taken into consideration in determining operability. I have frequently observed, especially when the focus was in the kidney, testicle or epididymis, that the spinal lesion went on to a successful issue following operation, although the general resistance of the patient was insufficient to prevent the progress of the second focus or foci either to the point of destruction of the affected organ or to the necessity for its extirpation. I have observed the same favorable result after operations on monarticular joints in the face of tuberculosis elsewhere. The presence of a second tuberculous focus, even if it is progressive, does not contraindicate the operation.

(3) *Field of Operation.* The presence of sinuses is not a formidable obstacle unless they are situated near the spinous process or in the field of operation. Fortunately, the anatomic planes of cleavage are so placed that it is rare for a cold abscess, or ichor pocket to point in the region of the spinous processes. They may point close enough in the loin to the field of operation to demand special care. The skin must be thoroughly prepared and the sinus sealed with a collodion tampon before the operation is begun. The indication for operation is increased when an abscess or sinus is present.

In 4 cases I have unexpectedly encountered an ichor pocket deep in between the spinous processes. In none of these did it interfere with the union of the graft or the course of convalescence.

(4) *Age of the Patient.* The bone-graft operation has been more readily accepted

by the profession in adults than in children. It has been held that, since the bones in a child are smaller and not fully developed, the operation should be postponed. My position is that advantage should be taken of the operation to afford the child the aid of ankylosis at the earliest possible moment.

In a case of acute progressive spinal disease in the child, one often observes excessive motion at the center of the kyphosis accompanying respiratory movements. This indicates that the underlying vertebral bodies have disintegrated; the respiratory thrust has been concentrated into the area left without the immobilizing support of the intact vertebral bodies. A vicious circle is set up: the greater the mobility, the more rapid the destruction; and destruction increases mobility. The bodies melt away progressively.

There is no room for argument in such cases. It is clear that the process must be checked as soon as possible. It is equally clear that frames and braces cannot be expected to meet such demands. Operative treatment is decidedly indicated.

If a continuous bone-graft bridge is inserted at the point of greatest mechanical advantage, namely, the distal portion of the spinous processes, the bony elements at the site of the kyphosis, instead of being weakened, are supported by strong new bone with maximal leverage; and the minute the sutures are tied, immobility is favorably influenced.

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PSYCHONEUROSES

SECONDARY TO HEAD INJURIES

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IN a series of some 500 cases of head injury studied by one of us (M. A. G.), it was quite evident that many of these patients developed psychoneuroses.[1] These psychoneuroses, although indirectly due to the head injury, were extremely disabling factors and frequently led to lengthy and in some instances permanent total disabilities. By the realization that such psychoneuroses develop quite readily in patients suffering from head injury one should be ready to combat this development. Although much is known and accepted in the treatment of head injuries along neurosurgical lines, it appears that relatively little attention has been devoted to the psychiatric phases. We, therefore, direct consideration to certain principles of treatment derived from investigation of the motivating factors influencing human behavior.

It has been our experience that maladjustments in the emotional realms (finances, family difficulty, antagonism to physicians, etc.) are of great importance. Certainly patients must sense the continued support of the physician and his genuine interest in them. By such personal interest and attempts at readjustment many cases have been restored to a wage-earning basis and complaints comparatively readily resolved. We must emphasize that such a clear-cut development occurs in only an uncertain proportion of cases. In many patients the differentiation is difficult or impossible, but a sufficient number of psychoneuroses do develop on a basis of such situations as to warrant investigation of the subject. In brief, these types of maladjusted situations are: (1) antagonism to physicians in charge; (2) financial difficulties in relation with insurance carriers; (3) difficulties in home life relationships. It must be further borne in mind that our point of view does not involve treatment at a "deep," or psychoanalytical, level.

In many of the cases there is definite brain damage ranging from slight to severe. This is evidenced by the presence of organic neurological findings, such as hemiplegia, cranial nerve injuries, *x*-ray evidence of fracture, abnormal vestibular findings, bloody cerebrospinal fluid, and definite pathological findings by encephalography. How long these symptoms referable to organic damage continue it is impossible to state, but it does seem certain that head injuries do something more; they produce a "sensitization" favorable to the development of psychoneurotic symptoms. In time these complaints come into the foreground and become the incapacitating ones for the patient. These psychoneurotic findings consist of such symptoms as nervousness; headaches becoming variable in location, character, and frequency; dizziness, originally clear cut, becoming changeable and bizarre; body pains; fatigue and mood changes. Associated with these may be the objective findings of vasomotor instability, tremors, variable reflexes, not indicative of an organic lesion, etc.

To explain this state of affairs requires some resort to the concepts of behavior motivation. There exists without doubt in all of us a very strong tendency to evade responsibility and the assumption of adult behavior requirements. It is vastly easier to follow the route of minimal effort. But the demands of a social situation are such as to compel the assumption of such adult

[1] Skull and brain traumas: their sequelae. *J. A. M. A.* 98: 271–275, 1932.

responsibilities. Indeed, in the usual instance, the pressure of this social demand has been so insistent and so long in duration as to result in an adaptation that is quite efficient. These demands cease to operate from outside the individual and are incorporated into his own personality makeup. In other words, these requirements have been internalized, so that they operate automatically and more or less unconsciously from *within*. This process is really nothing more or less than the learning process of experience, for it has long been recognized that to have meaning an experience must be made an integral part of the individual.

But underneath all this socially adaptive learning remains the unconscious desire to avoid responsibilities of making a living, difficult decisions, etc. To gravitate towards a more infantile state, where service is obtained without paying the attendant price is a tendency requiring a strong counter-balance. When the equilibrium is seriously disturbed this tendency is given an opportunity to express itself.

Now, so long as a reasonably adequate physical status exists, this tendency is effectively inhibited, because to manifest itself would jeopardize the internalized social standing of the individual with himself. He could not square it with his sense of right; to yield to the tendency would be to invite conscious guilt feelings. But given a definite physical injury the self-responsibility is avoided. It is now shifted onto something outside of the self, i.e., on the physical body. It has been generally accepted that a physical disability is a justification for adjusting at less mature levels than is otherwise expected. The incurring of a physical disability, then, has the potentiality of substituting for the sense of guilt that would otherwise operate, and releasing trends ordinarily well masked by the development of a strong social conscience. *This occurs without conscious realization by the patient.* He is therefore quite completely honest (so far as he knows) in stating his complaints and in assigning the injury as causal. In fact in this indirect but none the less real sense, the injury was the cause. But the continuance of the symptom is now of some value to the patient; he is able to drop to less strenuous planes of activity and at the same time does not have to yield any of his self-respect in so doing. He is absolutely sincere in the expression of a desire to regain his health; but unknown to him he has a powerful urge to remain disabled.

It is the viewpoint outlined in the foregoing discussion that enables us to understand the development of the psychoneurotic syndrome superimposed on the true organic one. Unless this is borne in mind the therapeutic endeavors aimed effectively at the original trauma may, by their continuance, contribute to the development and perpetuation of the superimposed psychoneurosis. It becomes necessary to consider almost from the outset the total personality of the patient, his adjustments in the various fields of emotional experience.

It is of course possible to classify head injury cases in almost innumerable ways. For our purpose the following appears a workable one, being based upon the subsequent psycho-emotional reaction to the injury:

(1) Head injuries that progress rapidly either to fatal termination, or in which the patient recovers and returns to work without the development of neuroses.

(2) Head injuries that develop neuroses; the presence of this is recognized by the attending physician; prompt measures along constructive lines are instituted, and the patients recover.

(3) Head injuries that develop into full-blown psychoneuroses, but in which, under appropriate treatment, the patients make a satisfactory adjustment and return to duty.

(4) Head injuries in which psychoneuroses develop and in spite of all efforts or because of inadequate efforts, continue as disabling syndromes.

The following case presentations are chosen from the series of 500 head injuries

and represent types falling more or less naturally into this scheme of classification. They are simply instances in which certain features of the psychiatric point of view are found applicable. Obviously the first type, in which neurosis is not a factor, does not concern us here.

The second type, wherein the neurotic implications are early recognized and considered, is illustrated in Case 1.

Case 1. P. B., aged thirty-five, fell 12 feet, striking the back of his head on a steel rail. He was semi-conscious, with hemorrhages from both ears. He was in shock. There was fracture of the left clavicle and left facial palsy. Following recovery from shock, dehydration measures were instituted. X-ray showed no fracture. There was great improvement in the neurological findings and he was discharged from the hospital in about four weeks. Neurological examination at that time, however, showed some left facial paralysis and loss of hearing in the left ear. About that time he began to develop attacks of dizziness lasting for about a minute, upon arising and lying down. He began to complain of trivial aches and pains in the body, became nagging and petulant, exaggerated his complaints, with every day a new one. He was taken from the hospital to his home, where for a time he was nervous and irritable. His family situation was fairly good. Compensation was set on a basis of eighty dollars, comparing with his former wage of one hundred dollars. His complaints were sympathetically received, but he was encouraged constantly to return to light work sooner than would ordinarily occur in such cases. He gradually was induced to return to complete work and has continued on this.

Here was a case with severe head injury. Had he been treated strictly on the basis of the trauma he would very probably have been hospitalized sufficiently long to "fix" his neurotic developments. But looking to the emotional aspects of his problem and making it worthwhile so far as possible for him to return to work, motivation was enlisted in this favorable or constructive direction instead of in the destructive direction of increasing symptomatology.

Type 3 is perhaps illustrated by Case 11.

Case 11. T. S., aged thirty, was struck on the back of the head by a heavy plank. He was unconscious for fifteen minutes and after a brief return to consciousness lapsed into a stupor for eleven hours. There was slight bleeding from the nose, mouth and left ear, and paralysis of the left face and left lateral rectus. X-ray showed fracture of the left parietal bone. He was hospitalized for two and one-half weeks and then cared for at home for about three months. Following the injury he developed attacks of headache lasting three-quarters of an hour and occurring several times daily, also unsteadiness, particularly in elevators and high places. He complained of blurred vision, fatigue, extreme nervousness, was easily frightened, showed marked changes in disposition, becoming quarrelsome, irritable, and at times moody, sad, and desirous of being alone.

The patient had considerable domestic difficulty. Becoming involved in a fistic encounter, he became responsible for costs having nothing to do with the original injury. He purchased an auto on his veteran's bonus, and straight-way wrecked it. He became involved in a love affair necessitating financial assistance. During this entire time his symptoms persisted. In fact the more financially involved he became, the worse were his symptoms.

It was decided to recommend financial settlement. $350 in total settlement was given, with the recommendation that he spend some time on a ranch. Six weeks later he returned, greatly improved. Two years later he still reports good condition, with continuance at work.

Here was a bona fide head injury followed by neurotic symptoms. These were accentuated by home difficulties and lack of funds. A lump sum settlement entirely stopped the progress of these symptoms. It seemed as though the final responsibility for his actions was now irrevocably thrust before him in ways he could not evade.

The following is a case where psychoneurotic symptoms were noted in incipiency, but in which the advice given for therapy was refused, with the result that within nine months the patient was totally and apparently permanently disabled. This corresponds to Type 4 of our scheme:

Case 111. J. A., aged thirty-eight, was struck in the suboccipital region and spine by

a heavy rock. He was unconscious for a few minutes, with a dazed condition for several hours. There was bleeding from both ears, and fracture of the 5th spinous process. He was hospitalized for two days, followed by eight days at home. He was then returned to work on a full time light duty basis. Because of the general economic situation he was discharged within a month. He then developed continuous headaches, dizziness, fatigue, insomnia, and became quarrelsome and irritable. His family consisted of a wife and three children. It was the first time in his life that he had found himself unable to meet his financial responsibilities. The insurance company refused compensation, whereas at that time he could have been placed on partial disability and given some light work. Within two months he was unable to do any work and had to be placed on a total disability. His symptoms have steadily increased until now, one year later, he is unable to do any type of work.

It would seem that if this man could have been kept at light duty, with continuance of partial compensation permitting an extrication from his debts something could have been accomplished in rehabilitation.

Quite as a matter of course, our point of view implies no combat with the usual neurosurgical measures of treatment. These accepted methods of dehydration, surgery if indicated, sedatives, physiotherapy are adequately dealt with in numerous articles on acute head injuries.

In Types 2 and 3, in which some measure of results has been obtained, treatment has been directed not only along these recognized lines, but on the basis, as well, of the principles already outlined.

When we reach Type 4, with fixation of psychoneurosis, often a more superficial level etiology is not discovered. Presumably a deeper level or semi-analytic therapy is required in these instances. Inasmuch as analytic therapy, with its tremendous expenditure of time and money, is impracticable in the vast majority of cases, it need not be considered here. But if cases are watched carefully by the attending neurosurgeon, with detection of the neurotic developments in their incipiency, it is our feeling that relatively smaller numbers will reach this fourth type.

CONCLUSIONS

Head injuries associated with organic cerebral pathology are prone to develop a superimposed psychoneurosis, which may become even more disabling than the original damage. This point of view is based upon the modern dynamic concepts of human motivation. With the realization of this point of view and its incorporation into actual clinical practice, it is our belief that the therapy of head injuries can be augmented and many late sequelae minimized. In cases of well-developed psychoneuroses, something approaching psychoanalytic therapy may be necessary, but it may be possible to prevent as large a number as at present from reaching this stage.

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FRACTURES OF THE CARPAL NAVICULAR AND TRIQUETRUM BONES

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THE interest in fractures of the carpus since the advent of *x*-ray has more or less centered about the navicular bone, mainly because it has revealed the frequency with which this structure is involved, and has enabled a positive diagnosis of fracture to be made where formerly the condition might have been classed as a simple sprain.

Fractures of the remaining bones composing the carpus, other than the lunate, have received comparatively little attention in the literature. Sporadic cases have been reported, but because of their rarity no lasting interest has been stimulated.

During the past year, in the Out-Patient Department of the Roosevelt Hospital, 6 cases of fracture involving the os triquetrum, and 15 cases of fractured navicular, were diagnosed and treated.

No other bones of the carpus were involved in this series, and because in 2 of our cases, both navicular and triquetrum were fractured in the one wrist, it was felt worth while to discuss both conditions at the same time.

ANATOMICAL CONSIDERATION

The wrist joint proper is formed by the articulation of the lower articular surface of radius and the discus articularis, with the proximal row of carpal bones; the os naviculare, lunatum and triquetrum, with the pisiform which lies in intimate contact with the volar surface of the latter bone. Its proximal articular surface is concave, both anteroposteriorly and laterally, making it a condyloid type of joint, allowing much flexibility and making circumduction of the hand at the wrist possible.

With the hand held in the long axis of the forearm, the greater portion of the radius is in contact with the proximal two-thirds of the navicular bone, while its ulnar third and the discus articularis are in contact with the lunate, the os triquetrum entering into the articulation only when the hand is adducted to the ulnar side.

Of lesser importance from the point of view of this paper are the distal row of carpal bones, which articulate with the proximal row to form an irregular undulating joint line, the os hamatum and os capitatum forming a high convexity in the proximal direction, while the proximal surfaces of the os multangulum majus and minus lie more distally. This is obviously an arrangement which does not permit of lateral movement, so that in lateral motions at the wrist joint they move in unison with the proximal row.

In visualizing the ligamentous arrangement of this joint, imagine a common capsular ligament extending from the articular borders of the radius and ulna to the bases of the metacarpal bones. Between these attachments it effects communication with each row of carpal bones and in turn it is reinforced by four accessory bands, the dorsal and volar radiocarpal ligaments, whose main strength lies in the support of the overlying tendon sheaths, and the two lateral ligaments which are most important. These ligaments are called radial and ulnar collateral respectively. The former passes from the radial styloid process to the base of the first metacarpal bone, and in addition is attached to the tip of the navicular and the os multangulum majus, while the latter passes from the tip of the ulnar styloid process to the base of the fifth metacarpal and establishes similar communications with the triquetrum and hamatum bones.

It must be remembered that accessory

centers of ossification can occur in the navicular bone, and for this reason one must not confuse a bipartite condition with a fracture.

INCIDENCE

It is unanimously agreed that carpal injuries are seen most frequently in the male. Our series, which shows 16 occurring in men, and 3 in women, with the ages ranging from thirteen to forty-eight years, and 80 per cent of these occurring under the age of thirty-five, bears out this statement.

We have been unusually successful in collecting a total of 21 cases of fractured carpal bones during the one year period from July, 1931 to July, 1932. Sixteen were seen in the Out-Patient Department and 5 in the Compensation Clinic. Of this total, 15 showed fracture of the navicular bone, 2 showed a combination of fractured navicular and fractured os triquetrum; while 4 showed fracture of the os triquetrum alone. The unusually high percentage of fractures of the latter bone, in our series, can in part be explained as mere coincidence, as is the fact that we have no single instance of injuries to any of the carpal bones other than navicular and triquetrum.

By careful search through the literature a moderate number of isolated cases of fractured os triquetrum can be found. In each instance the author has been impressed by its rarity and the comparative lack of material concerning its existence as a fracture entity. No one person has gone into the subject at length for the simple reason that none of them has personally seen more than a few cases. Our collection of 6 cases within a one year period would suggest that these fractures are more prevalent than the statistics would imply. The reason can be found in the fact that the pisiform usually obscures the fracture line in the anteroposterior *x*-ray view, and the average lateral view places three bones: the navicular, lunate and the head of the capitate, in the pathological line to obscure visualization further.

It is suggested that when a fracture of this bone is suspected, an oblique view be taken, to place the triquetrum in a position where it is not obscured by the other bones.

In 2 of our cases a simple anteroposterior view might have been passed as negative, for the pisiform partially or completely obscured the fracture. A lateral *x*-ray taken in the foregoing manner made the state of affairs evident.

PATHOLOGY

Os Naviculare. In fresh fractures of the navicular we see the following varieties:

1. *Fracture of Tuberosity* (see Figs. 2A and B): Here that portion of the tuberosity which gives attachment to the radial collateral ligament is evulsed or incompletely fractured. It may vary considerably in size and extent, and is produced by any motion or force at the wrist which will put this attachment on sufficient strain. All of our cases gave a history of fall onto the palm of the dorsi-flexed hand, and I feel that it was produced in combination with direct force and a twisting motion which will be explained later.

These fractures are not intra-articular, but within the ligament, showing no hemarthrosis. The blood supply to the bone is not materially damaged and because they are usually incomplete fractures they heal rapidly and bony union can invariably be expected in spite of the treatment employed, but, because of the initial extreme pain and discomfort, it is wise to keep them in splints for several weeks.

2. *Transverse Fracture through Neck or Body of Bone* (See Figs. 1 A and B, 3, 4, 7 A and B): This is the type where the plane of fracture is at right angles to the long axis of the bone. It should be divided into two groups:

(a) Incomplete: Here the fracture line which begins on the proximal articular surface of the bone does not involve its entire thickness, but presents the green-stick appearance. Age seemed to have no

bearing on its existence.

(b) Complete: The heavy fracture line in this variety can be clearly visualized extending across the entire thickness of bone. There is little tendency for displacement of the fragments unless an associated dislocation of the lunate exists.

The exact position of the fracture varies to a slight degree depending upon the individual shape of the navicular, but it is at the weakest and, usually, the narrowest portion of the bone, often described as the neck. In an overwhelming majority of cases the fracture can be accurately placed at a point representing the junction of the proximal third with the distal two-thirds of the bone (shown nicely in Fig. 7A) and lies definitely distal to that portion of the navicular which is intimately embraced between the head of capitate and the articular surface of the radius.

FIG. 1 A. J. H., Feb. 27, 1932. This illustrates type which we have described as incomplete transverse fracture of navicular bone; it occurred in equal frequency to complete transverse type, each having an incidence of 25 per cent in our first 20 cases of fractures of carpal bones. This type should rarely show non-union in comparison to those in which crack is seen extending across entire body of bone.

FIG. 1 B. Same patient, April 5, 1932. Check-up *x*-ray seven weeks after original injury, showing complete bony union.

There is, of course, a local embarrassment of blood supply along the fractured surface of each fragment due to the crushing of spongiosa, but relatively the distal fragment is the better nourished of the two, the main source of blood apparently reaching the bone via the ligamentous attachment to its tuberosity, and to a lesser extent via its thin periosteal covering. This fracture, being intra-articular, results in varying amounts of hemarthrosis.

If complete immobilization is effected, new blood vessels bridge the gap from the healthy distal half to the undernourished proximal portion and union results. Otherwise a slow absorption of bone occurs along the fractured surfaces and gradually the plane of fracture widens and cavitation into each portion may take

place, this osteolysis undoubtedly being promoted by the abnormal motion between the severed edges.

a foreign body within the joint. Speed's theory is to be questioned following the findings of R. V. Grace, who has had

Fig. 2 A. Fracture of tuberosity of navicular, type that shows nicely in anteroposterior view; but one must remember that extreme tip which projects volarward may be only portion involved, and so could most easily be demonstrated by oblique lateral view.

Fig. 2 B. Check-up *x*-ray taken at end of three weeks which shows bony union even though former fracture line can be faintly detected.

The later picture, as seen in old untreated cases, usually shows the distal fragment presenting atrophy from disuse, with perhaps mottling as though from replacement fibrosis, while the proximal fragment does not show this change. As explained by Kellogg Speed, this indicates that all the blood supply of the proximal fragment is cut off, it becomes dead, and presents an increasing opacity with time to the *x*-ray, while the distal fragment, having retained some of its blood supply, begins to show the atrophy of disuse. Our one case (Fig. 3), where excision of the whole bone was performed, showed the foregoing changes in the preoperative *x*-ray, and in addition the proximal portion appeared as a separate bone with smooth, well-rounded surfaces, evidently lying as several operative cases in which microscopical examination of the fragments revealed degenerative changes involving both fragments.

The particular physiological state of the proximal fragment where the fractured surface, by *x*-ray, has become well rounded off and no longer presents a rough surface to which union might take place, seems to represent one particular phase in the evolution of non-union where operative removal would be the only choice, should the symptoms be of sufficient severity to warrant the step. Up to this point treatment by immobilization should be given a chance, as shown by the work of Böhler who has union occur even six months to a year after the original injury. In very old cases osteoarthritic changes occur in the region of the navicular which may result

in varying degrees of limitation of movement, even to ankylosis of the wrist in its midposition.

2. Chip fractures of the dorsum of the bone. In this group we had 3 cases.

3. Chip fractures involving an angle of the bone. In this group we had one case involving the proximal internal angle of the bone.

Fig. 3. Preoperative *x*-ray in only chronic operative fracture in our series. Arrow points to clearly defined proximal fragment, which shows evidence of increased calcification while distal fragment presents more mottled appearance. Notice clearer proximal row, due to greater retention of calcium salts, an probably caused by fact that in this injured wrist very little functional movement was occurring between the two rows, but instead between proximal row and articular surface of radius.

Fig. 4. G. A. April 20, 1932. Showing combination of transverse fracture of navicular and chip fracture of proximal angle of os triquetrum. Arrow points to fracture line which does not stand out as clearly in print as it does in *x*-ray film.

Os Triquetrum. In fractures of the os triquetrum (see Figs. 4, 5, 6) the bone may be chipped at one of its angles or on its dorsum but the usual site of fracture is reported as its midportion transverse to the long axis of the bone. It may be a single cleft or may be shattered into many fragments by means of the force transmitted through the pisiform. These fractures should heal rapidly with adequate immobilization.

In our 6 cases we could divide them into the following pathological types:

1. Transverse fractures of the body. In this group we had 2 cases.

The transverse fractures were clean cut and showed a definite, though slight, separation of the fragments. The pisiform in each instance was completely superimposed in the anteroposterior view, necessarily confusing the picture unless the observer is experienced in interpreting *x*-rays of the carpus. An oblique view in one of these cases made the pathological condition much more apparent. (Figs. 5 A and B illustrate this nicely.)

The chip fractures in our series predominated by two to one, and showed a predilection for the dorsum of the bone. A straight lateral view in each case showed the pathological condition but an oblique

view was necessary to rule out sesamoid bones and possible extraneous calcifications.

The mechanism in cases of indirect violence is more complex, it is applied to the bone involved through the medium

Fig 5a. Fig 5b. Fig. 5c.

Fig. 5 a. A. K. Jan. 8, 1932. Arrow points to region of pathological condition showing transverse fracture of os triquetrum underlying pisiform. Compare Figure 5 b which shows how much more clearly one can visualize this fracture by employing oblique lateral view.

Fig. 5 b. Same patient. Oblique lateral view, which exposes fracture line unobscured by dorsal portions of navicular and lunate bones; which would otherwise make visualization extremely difficult in those cases of fractured os triquetrum. Arrow points to fracture and one can detect what seems to be very slight distal displacement of inner fragment; outer fragment being stabilized by ulnar collateral ligament of wrist joint.

Fig. 5 c. Same patient. June 14, 1932. Check-up *x*-ray five months after injury, fracture line can stil be detected but there is evidence of early bony union.

"Mechanism of Fractures"

We may consider as the cause of these carpal fractures both direct and indirect violence; although direct violence is rarely an actual cause in fracture of the navicular bone, it plays a prominent part in injuries to the os triquetrum.

The mechanism in cases of direct violence is easy to visualize, it needs no explanation. There is external violence applied directly to the bone involved, perhaps through the medium of the pisiform as in triquetrum fractures; it may be by the blow of a hammer, or the carpus may be crushed between two objects. The force here is applied directly to the bone involved, and the extent of damage depends directly upon the intensity of the force.

of other structures, and in a manner not so easy to comprehend.

Considering indirect violence as the usual cause of a navicular fracture, we find that this can occur:

1. Where the forearm and hand are quiet.

2. Where the forearm and hand are moving parts.

Under the first group would come the injury to the chauffeur's wrist when the car backfires, or the steering wheel suddenly jerks back; or any similar type of injury, where the forearm and hand are not in motion. The usual result in these cases is a fracture of the radial styloid process, but do not forget that the carpal bone may also be involved.

Under the second classification come

falls on the hand either forward or backward, with the palm down and very rarely on the back of the hand. In falls forward on the palm the hand is usually very slightly deviated radially although the deviation may be to the ulnar side, while in falls backward onto the palm, the hand is almost constantly deviated radially. The hand, in addition, is usually dorsiflexed to an angle of about 45° with the forearm; with the weight of the body as it falls being resisted by the immovable hand, the force is transmitted from hand through wrist to forearm. This position is usually that of slight radial deviation, with dorsiflexion; therefore it can be supposed that the navicular with its neighboring proximal row of carpal bones glides ulnarward at the same time that it rotates dorsalward. A more important factor, however, is that synchronous with the impact of the fall or violence, whatever be its exact nature, there is an exaggeration of dorsiflexion beyond that limit set down by nature.

Fig. 6A. Fig. 6B.

Fig. 6 A. Os triquetrum is partially outlined and an arrow indicates chip fracture upon its dorsum. An arrow also points to an incomplete fracture on volar aspect of radius. (Again print does not show pathological condition as clearly as *x*-ray.)

Fig. 6 B. Check-up *x*-ray after five months displays dorsal chip not completely united.

Most explanations of the mechanism involved in transverse fractures of the navicular bone rather glibly infer that the force passes through the head of the capitate and is transmitted directly to the navicular, impinging it against the articular surface of the radius and causing it to fracture at this point.

In the great majority of instances it will be observed that the head of the capitate comes into intimate contact only with the medial portion of the proximal fragment, some distance removed from the actual fracture line. For this reason it seems that some other explanation should be offered as to the cause except in the rare instance where the fracture is seen extending across the innermost portion of the bone.

Following this line of thought a special *x*-ray (Fig. 7C) was taken of one of our patients who already showed bony union of a previously fractured navicular, and had perfect function as compared to his normal wrist. From this *x*-ray we established the interesting observation that the hand in a position of extreme radial deviation and dorsiflexion shows the navicular to have glided ulnarward barely a quarter of an inch from that position which it occupies with the hand in the long axis of the forearm, and that the former fracture line is still on a level distal to the radial styloid. It was also noted that the os multangulum majus had rotated onto the dorsal surface of the tuberosity of the navicular, and was grossly simulating an extent of gliding ulnarward which actually did not exist. In addition it could be observed that otherwise the distal row of carpal bones did not change in its relation to the proximal row.

On the basis of these findings I wish to propose the following theory of the mechanism in fractures of the navicular bone.

It is my belief that during the motion of extreme dorsiflexion the tuberosity of the navicular, being attached to the strong collateral ligament and stabilized by the os multangulum majus, which has rotated onto its dorsum, offers resistance to the rotary force applied to its mobile proximal portion by the head of the capitate. The effect is a twisting violence applied to the navicular bone, and if it be sufficient the bone fractures at its weakest portion.

Fig. 7A. Fig. 7B. Fig. 7C.

Fig. 7 A. W. N. March 15, 1932. Type illustrating complete transverse fracture of body of navicular. It is in this type particularly that one should be on lookout for non-union.

Fig. 7 B. Same patient. June 14, 1932. Check-up *x*-ray taken three months later shows bony union; on very careful inspection it is possible to detect a faint line representing site of former fracture.

Fig. 7 C. Same patient. June 14, 1932. This illustrates interesting point brought out under "mechanism of fractures" that extreme radial deviation and dorsiflexion do not result in any appreciable lateral gliding of navicular along articular surface of radius, as can be seen by comparing Figure 7 B. It also shows how multangulum majus and minus have rotated to its dorsal and lateral aspects, more or less embracing its tuberosity and acting as a stabilizing factor.

The anatomy of the wrist, the presence of transverse green-stick fractures involving only the proximal surface of the bone, and the usual situation of the fracture well lateral to the plane of most intense force, would seem to substantiate this theory, as would the extreme rarity of fractures involving the lunate bone or medial third of the navicular, which certainly come into more intimate contact with the head of the capitate.

The same twisting force would explain the evulsion of a navicular tuberosity as seen in several of our cases, and would account for the severe localized traumatism to the radial collateral ligament seen in bad sprains, where the intense pain and disability of the wrist with all the cardinal signs of a navicular injury give an *x*-ray negative for fracture. In the latter instance one must presuppose that the multangulum majus had not rotated to that point on the dorsum of tuberosity where it could act as an additional bracing factor, the radial collateral ligament, as well as the tuberosity, receiving the entire expenditure of force, and either one giving way.

In a search through the literature it has been difficult to obtain any very expansive views as to the mechanism producing fracture of the os triquetrum. Kellogg Speed, in

his excellent monograph on traumatic injuries of the carpus, points out that a few cases have been reported and that he

Fig. 8. E. S. Oct. 23, 1932. Combination of transverse fractures of both navicular and triquetrum bones. Proximal border of pisiform bone is seen just overlying fracture of triquetrum. Pathological condition is much more evident in actual *x*-ray film. Arrow points to fracture level.

feels they are caused by direct crushing injury to the bone with the hand in ulnar deviation.

In reviewing our cases a direct injury to the bone through the medium of the pisiform seems the most logical explanation for the transverse fracture. In both instances the weight of fall having been received on the palm. One patient in whom the navicular was also transversely fractured (Fig. 8), fell with the handle of a hammer clutched in his palm; he must have sustained the fractured navicular by the twisting violence, and the fractured triquetrum by direct force through the pisiform. Of the 3 cases in which the dorsum of the bone was chipped, two gave definite histories of falls onto the back of wrist, while the third could give no definite account of how his fall had been sustained.

Several cases have been reported where the ulnar portion of the wrist has been severely crushed between two objects, accounting for the fracture.

SIGNS AND SYMPTOMS

In navicular injuries the symptoms necessarily vary with the freshness of the injury and for this reason it is well to divide them into acute and chronic cases. The most striking feature in the acute case is the original loss of function, the patient carrying his hand and wrist well splinted in a protective manner. There is a moderate amount of swelling, more or less limited to the carpus, with an obliteration of the anatomical snuff-box. The maximum point of tenderness is well localized over the dorsal and lateral aspects of the navicular bone. Axial pressure along the third metacarpal, with the hand held in radial deviation at the wrist, could not be relied upon, at times being present when the fracture was absent and vice versa; this was also an impression gained by Grace in his splendid group of cases presented in 1929 before the New York Surgical Society. All motions at the wrist were painfully limited, probably dorsiflexion and radial deviation to the greatest extent. In those cases which showed only a fracture of the tip of the tuberosity, there was often only localized tenderness over the volar aspect of the wrist, overlying the point of fracture.

In the chronic cases there is often a slight fullness and definite tenderness in the anatomical snuff-box, and to a lesser extent pain on deep pressure over the dorsal aspect of the bone. Dorsiflexion and radial deviation are definitely limited and extremely painful when forced. The wrist and forearm show varying degrees of muscular atrophy, depending upon the length of time which has intervened since the original injury. This atrophy, however, is a usual feature, if sufficient time has elapsed.

Our cases of transverse fracture of the os

triquetrum were all acute, showed definite swelling over the carpus, painful limitation of all movements at the wrist, particularly ulnar and radial deviation. Deep palpation definitely localized the tenderness to the dorsal aspect of the bone which is situated about half an inch distal to the ulnar styloid, and by means of pressure transmitted through the pisiform would localize tenderness to the same area.

Those cases in which the bone was chipped on its dorsum showed a moderate amount of swelling involving the carpus, were excruciatingly tender over the fracture site, with pain referred to the dorsum of the bone on forced dorsiflexion; and a similar syndrome on heavy palmar pressure. The loss of function varied directly with the extent of injury and the amount of swelling. All cases showed a definite loss of grip.

PROGNOSIS

In fractures of the tuberosity and in incomplete fractures of the body of the navicular the prognosis is unfailingly good; while in complete fractures through the neck or body it must be guarded, depending to a great extent on treatment. If they are diagnosed and treated early by immobilization in a cock-up splint, until a check-up *x*-ray shows bony union, the result should be excellent. This is borne out by our own experience in which we have had no single case of non-union.

Unfortunately, or fortunately, we have only 2 cases in which diagnosis was made late. One is still in his first four weeks of immobilization and I plan on keeping him thus for six weeks longer, or until a check-up *x*-ray shows definite union. His injury occurred about six months ago, and when first seen by us the *x*-ray showed beginning cavitation in both fragments, and there is no reason why the end-result should not be entirely satisfactory.

In those cases where operative removal of either proximal fragment or entire navicular bone is performed (the procedure that was followed in our first chronic case), the results depend entirely upon the extent of osteoarthritic involvement of the joint that has already taken place; as well as the atrophy from disuse which has permanently affected the muscles and ligaments that are concerned in the function of the joint. In general, it can be said that the functional results after excision in those diagnosed very late are poor; but the pain is usually relieved and should be the indication for operation.

The prognosis in fractures of the os triquetrum should be good. Two of our 6 patients have been lost track of, and cannot be traced, but they were discharged symptom-free. Of the remaining 4, one has a perfectly normal wrist in every respect, 2 are symptomless except for a referred pain to the dorsum of the bone when transmitting heavy palmar pressure through the pisiform bone, while the fourth has only a very slight limitation of dorsal flexion. It was possible to get a check-up *x*-ray after three months in only one of our cases of transverse fracture of the os triquetrum, and although there was considerable filling in of the fracture line, it presented much the same appearance as a fibrous union. The wrist function is perfect and the patient complains only of occasional pain over the fracture site on heavy palmar pressure.

DIAGNOSIS

The diagnosis can very frequently be made on the physical findings but the signs so closely resemble those seen in severe sprains of the wrist that *x*-ray examination is the only means of reaching an immediate and definite decision.

If an *x*-ray photograph were taken of every sprained wrist it would be surprising to find what a very large number of them are really cases of fracture. Although I have not *x*-rayed quite all of the sprained wrists seen in the Out-Patient Department during the past year, I have made the very interesting observation that 38 per cent of our sprained wrists showed fractures of the carpal bones. Before accepting this state-

ment literally it must be borne in mind that most of those sprains in which the patient returned to the clinic for further observation were of the severe type, so that my percentage calculation represents an exaggeration of the actual incidence.

It is most important when a fractured carpal bone is definitely suspected not to be discouraged by the ordinary anteroposterior and lateral *x*-ray views failing to display its existence; one must then take an oblique view, as in several of our cases this was the only angle at which the fracture could be visualized; this refers particularly to a fractured tuberosity of the navicular, and to fractures of the os triquetrum.

TREATMENT

Our whole series was treated by complete immobilization of the wrist in a cock-up position, by the use of a dorsal moulded plaster splint that extended from the midforearm to the level of the metacarpophalangeal joint. The thumb was not immobilized and our results justify the contention that any motion which may occur at the base of the thumb, under these conditions, will not disturb the process of repair. During the entire period of immobilization a special point was made to avoid manipulation of the wrist, even to the slightest extent; once a week the splint was carefully removed, the arm cleansed, and the splint refixed by means of adhesive strips, the whole being covered by a bandage.

The dorsal splint was used in preference to the volar one for several reasons: (1) The perspiration from the palm, particularly during warm weather, caused a softening of the plaster, an objectionable feature which was obviated by placing the splint on the dorsum. (2) It was found that with the wrist immobilized in this manner freer use to the fingers was permitted, and the patient could get real use of the hand. This usefulness made the patients protest less against the enforced period of disability, and, I feel, has kept the muscles and tissues of the palm in their normal healthy state, and has secondarily given a more adequate circulation of blood to the proximal row of carpal bones than would occur if the hand were put completely out of use.

You may be familiar with the congested boggy condition of the tissues sometimes seen in a leg which has been completely immobilized for a fractured tibia, and it is only logical to assume that the same unhealthy state of affairs exists deeper at the level of the fracture site; although one does not see this extensive gross change in carpal fractures, it is easy to appreciate that circulatory stasis would take place.

Johnson has shown experimentally in dogs that while bone repair does take place in the navicular, it is less active than in other bones. He found the zone of repair to be less extensive, and the fractured hyaline cartilage to heal by fibrous tissue. He showed there was no interference with the vascularity of the fragment which would cause either necrosis or interference with the hyperemia that might result in secondary delayed union or non-union.

This last point seems of greatest import though it does not indicate that the same pathological physiology should exist in the human. It throws doubt upon the claim of many that poor vascularity to the proximal fragment is the cause of non-union. Following this line of reasoning the additional good results that we have obtained by adequate immobilization would seem to favor abnormal motility of the fragments as the primary cause of pseudo-arthrosis.

The cock-up position was employed because it represents a position of optimum function in the hand, and by keeping the hand in extreme dorsiflexion from the start, we are assuring a maintenance of the particular motion so often limited as a result of these fractures.

The period of immobilization in fractures of the tuberosity varied from two to three weeks, depending on the size of the fragment. In transverse intra-articular

fractures the wrists were immobilized until a check-up *x*-ray showed definite bony union. This took anywhere from three to eight weeks, depending upon whether the fracture was incomplete or complete.

Our only chronic non-operative case is still in the fifth week of immobilization, but I plan on keeping it thus until the check-up *x*-ray shows definite union, even if it requires three months. Böhler says: "Put them up for six months if necessary," but of course that brings up the economic question which would deter most of us. One wonders, however, after hearing of some operative results, whether this lengthy period of immobilization would not justify Böhler's contention.

It is not the purpose of this paper to discuss operative treatment, mainly because of our paucity of material. Chronic cases with persistent pain are said to offer best results following operation, and opinion is divided between those who remove the proximal fragment and those who take out the whole bone.

The treatment of those in whom the os triquetrum was fractured, along with the navicular, was just the same as though the navicular alone had been injured; while in the isolated triquetrum fractures we shortened the period of immobilization to from four to six weeks, as by the end of this period our followed-up cases were symptom-free.

RESULTS

Those patients who completed our treatment, and in whose cases we were able to obtain a check-up *x*-ray are the only ones which are being taken into consideration. In considering results it is well to define the physical state which we have used as representing a cure:

(1) A check-up *x*-ray must show definite bony union.

(2) There must be sufficient functional return to insure no interference with the use of the part. This is particularly important in compensation cases.

(3) There must be complete absence of pain both when the part is at rest, and after heavy work; however we have overlooked the vague ache noticed so often during damp weather that is a frequent feature seen for months after most fractures.

(4) Complete absence of muscular atrophy.

The fractures will be considered in separate groups.

Table 1
ACUTE CASES

	Fractured Navicular			Fractured Triquetrum		Combined Fractured Navicular & Triquetrum
	Tuberosity	Incomplete Transverse	Complete Transverse	Complete Transverse	Chipped	
Number of cases	4	5	6	2	4	2
Number completing treatment	2	4	3	1	3	
Check-up *x*-ray obtainable	1	4	3	1	3	
Bony union by *x*-ray	1	4	3	1	2	
Non-union by *x*-ray	0	0	0	0	1	
Pain on discharge	0	0	0	1	1	
Loss of motion on discharge	0	0	0	0	0	

Acute Fractures:

1. *Navicular Tuberosity:* There were 4 cases, only 2 completed treatment and were discharged "symptom free"; a check-up *x*-ray was obtainable in only one case, this showed bony union. The average time of immobilization was three weeks.

2. *Incomplete Transverse Fractures of Navicular:* There were 5 of these cases; 4 patients completed treatment, in whom there were 4 cures by the aforementioned criteria.

3. *Complete Transverse Fractures of Navicular:* There were 6 in this group, but only 3 completed treatment, all of which were definite cures; of the other 3 one had a check-up *x*-ray and showed evidence of beginning bony union.

4. *Transverse Fractures of Os Triquetrum:* There were 2 cases, in only 1 of which treatment was completed. This man showed evidence of bony union, but complained of slight pain referred to the fracture on heavy palmar pressure.

5. *Chip Fractures of Os Triquetrum:* There were 4 such cases, but only 3 patients completed treatment; of these 3, one continues to have slight pain referred to the dorsum of the bone and a check-up *x*-ray after four months continues to show evidence of the chip fracture. The wrist in this case was immobilized only twelve days due to a dispute concerning the diagnosis. The other case shows bony union in the check-up *x*-ray after nine months and is a complete cure.

Chronic Fractures:

1. *Complete Transverse Fracture of Navicular:* We had only 2 of these, both diagnosed six months after injury. One of these patients underwent operation and presented the pathological condition previously described in the early part of the paper, the whole bone was removed and on a six months follow-up shows as yet no real improvement. The other case is still in its fifth week of immobilization and we plan on keeping it thus for three months if necessary.

Our follow-up on acute cases only represents about 40 per cent of the total, and our series covering a one-year period, although three times as large as any previous year, is nevertheless small. No single patient has returned to us complaining of pain or disability, in the group which did not complete treatment; and we have detected no single case of non-union in the acute group.

CONCLUSION

Fractured Naviculars:

1. Contrary to the general impression concerning complete transverse fractures of the navicular, the prognosis is good if they are diagnosed and treated early by adequate immobilization.

2. This adequate immobilization can be obtained, and will give the most satisfactory results, by employing a dorsal moulded cock-up plaster splint.

3. The period of immobilization should be two to four weeks for fractures of the tuberosities; four to six weeks for the incomplete transverse fracture, and six weeks to three months for complete transverse fractures.

4. Non-union is favored by abnormal friction between the fractured fragments, rather than embarrassment of blood supply to the proximal fragments.

Fractured Triquetrum:

1. These fractures are more prevalent than heretofore considered.

2. Visualization of the fracture can be aided through employing an oblique view.

3. It is not uncommon for the fracture to occur associated with a fractured navicular.

4. The prognosis is good.

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[For Remainder of References see p. 279.]

OSTEOCHONDRITIS DEFORMANS JUVENALIS OF THE OLECRANON*

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THE etiology of osteochondritis deformans juvenalis is still in a controversial state. Trauma,[1] infection,[2] and blood supply disturbances[1] are some of the more important and plausible theories of etiology put forward, no one of which has been conclusively proved to be the sole cause, but at the present time it may be said concerning the etiology of this interesting condition that the majority of students of the subject believe that in the affected individual there is a general predisposition to the condition, but some local factor, probably trauma, enters into the picture to bring about the actual lesion.

FIG. 1. Right elbow, lateral view. While photographic print of this radiograph does not show clearly fragmentation of olecranon epiphyseal bone center, irregularity of epiphysis is easily apparent.

FIG. 2. Right elbow, anteroposterior view. Trochlear epiphyseal bone center appears underdeveloped and fragmented in this view. This appearance is not considered definitely abnormal for age of patient.

It is the experience of those who have treated many cases of osteochondritis deformans juvenalis that the course of the condition is essentially unaffected by any treatment. The condition goes on to a determinable end result, regardless of what is done.[1] Osteochondritis deformans juvenalis was discovered first in the tibial tubercle ossification center by Osgood[3] of

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Boston in 1903 and independently by Schlatter[4] in the same year. In 1910, Legg[5] of Boston separated out a group of cases of hip joint disease which had been classified and treated as tuberculosis of the hip joint, but which had recovered with deformity of the femoral head and little damage to the hip joint itself. These cases were recognized to be a manifestation of osteochondritis deformans juvenalis. In the same year Calve[2] and Perthes[6] reported similar cases.

Fig. 3. Left elbow, lateral view. This photographic reproduction of radiograph of elbow shows fragmentation of olecranon epiphysis which is distinctly abnormal.

Similar lesions of the patella and of the tarsal scaphoid were reported by Koehler[7] in 1908. Sever[8] reported a similar lesion of the os calcis in 1912. Freiberg[9] reported a lesion of the second metatarsal head in 1914. Scheuerman[10] reported lesions in the primary centers of the spine in 1920 and Calve[11] reported lesions of the spinal epiphyses in 1925.

Fig. 4. Left elbow, anteroposterior view. Trochlear bone center of humerus in left elbow is better developed than center in right elbow and is not fragmented.

A survey of the literature on osteochondritis deformans juvenalis fails to show any reported cases of this condition in the olecranon.

Mandl[12] in 1922, considering the possibility of osteochondritis deformans juvenalis as a general rather than a local disease, radiographed various epiphyses in school boys having one clinical lesion. In his case reports, he reported one olecranon which he considered to be an asymptomatic lesion of osteochondritis deformans juvenalis but the findings in this case were neither clinically positive nor scientifically conclusive. Moreno[13] in 1928 reported a

case of a suppurative process in the region of the olecranon which resulted in the separation of the olecranon epiphysis entirely. This condition was an epiphyseal inflammatory lesion and a different clinical entity from the one under discussion. Panner[14] in 1929 reported an instance of a lesion in the capitellum humeri which was clinically and roentgenologically analogous to lesions of osteochondritis deformans juvenalis elsewhere.

The case which is here recorded is clinically and roentgenologically osteochondritis deformans juvenalis of the olecranon epiphysis.

A. E., a thirteen year old school boy, came to the New Haven Dispensary complaining of pain and limitation of motion in the right elbow. He ascribed his trouble to an injury five weeks previous when he was said to have been struck on the elbow by a baseball bat.

On examination, the right elbow presented no late signs of trauma, such as one would expect to see after such an injury as described. The boy was a well-developed and nourished child of normal size for the age given. He had marked tenderness on pressure over the right olecranon but no evidence of olecranon bursitis which might well give tenderness on pressure. The motion of the right elbow was limited in extension at 150° or 30° from normal by pain. Flexion of the right elbow was also painful at about 20° from full flexion. On examination of the left elbow for comparison, it was found that a similar condition existed except that the left elbow had only 20° of flexion deformity. The Kahn test was negative. The child was well in all other respects. Radiographs of both elbows were taken which showed fragmentation of the olecranon epiphyses. The roentgenologist, without knowing the clinical diagnosis which had been made in the case, returned a diagnosis of osteochondritis deformans juvenalis of the olecranon epiphysis.

In a study of the radiographs, it is interesting to note that the trochlear epiphysis of the humeri also shows some apparent fragmentation but this center of ossification is in such a tender age of development that this appearance might be considered within normal limits.

Under rest of the elbow, the sensitiveness and limitation of motion decreased but due to the uncooperativeness of the patient, the case could not be followed to a final end result.

The history of trauma in this case apparently had nothing to do with the production of the lesion since the findings in the left elbow were almost exactly the same as those in the right. Between the points of limitation of motion, the motion in the elbow joints was free from pain and no other signs characteristic of joint involvement presented.

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* Continued from p. 213.

A NEW METHOD OF REDUCING

FRACTURES OF THE HEAD OF THE TIBIA INVOLVING THE KNEE JOINT

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THIS method is one which, as far as I can determine, is new, and in view of the end-results obtained by this technique, it seems desirable to present the subject.

As we know from experience, many instances of fracture of the type shown in the accompanying *x*-rays, have been found difficult to handle and the results have not been satisfactory, not only in respect to faulty weight-bearing but also, in respect to limitation of motion in the joint proper, the patient usually being left with a permanently limited joint and a perceptible limp.

In my volume "Imperative Traumatic Surgery"* is shown a pair of knee splints which I include in this paper. At the time I worked out the splints for fractures into the knee joint I was obtaining unusually good results, but in some instances there would be some disturbance in the weight-bearing. For that reason I devised the following method, which I am now applying to all fractures of the tibia into the knee joint, with most gratifying results:

Professor Lorenz Böhler of Vienna constructed an implement called a *redresseur*, which is used primarily for the purpose of reducing deformities below the malleoli in fractures of the os calcis. I conceived the idea of having two large buttons of wood, 3½ inches long by 3 inches wide and 2½ inches thick, made of good solid wood such as oak, with a hole drilled on one side to fit the projection of the *redresseur*, and the other side concave to fit the contour of both sides of the knee.

In the vast majority of instances, it has been my experience that fractures occurring in and about the knee joint and involving the head of the tibia are usually located in the external tuberosity with an occasional fracture of the fibula in this vicinity. In these cases I wait for a week to ten days for the swelling to subside, then, under a general anesthetic, reduce the displaced fragments with this reconstructed *redresseur*, placing the fixed wooden button on the inside or normal area, and by manipulation with the handle of the instrument, producing forcible, yet steady pressure over the fractured area with the other button (see Figs. 1 and 2).

It has been gratifying to note the results of this type of steady pressure, as we know violence sufficient to cause a fracture of the head of the tibia usually produces a comminution of bone so that any attempt at forcible reduction by hammering, such as the method used by Cotton for hip fractures, is impractical and unsatisfactory. This is true because, with the amount of comminution, when one area is impacted and we then devote our attention to an adjacent area for a like purpose, we are very apt to break up the already impacted field. With this adjusted instrument which exerts steady pressure over the entire field, we get an equalized reduction which is much more satisfactory.

In the accompanying *x*-rays Figures 3–5 show a case in which I had not resorted to this new device, but had simply, by as much manipulation as possible, attempted a reduction, which, as is evident in the *x*-rays, was not satisfactory. Later I applied my original knee joint hinges. Remarkable to relate, even in this instance, with the knee joint hinges, this man has full extension and flexion, a well-controlled knee, no

* N. Y., Hoeber, 1929.

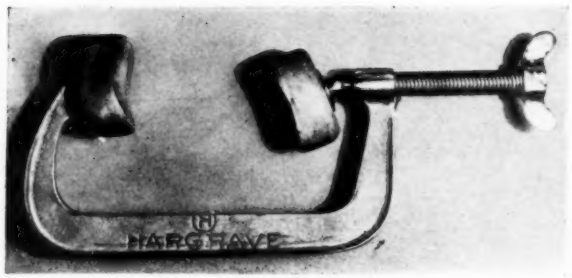

FIG. 1.

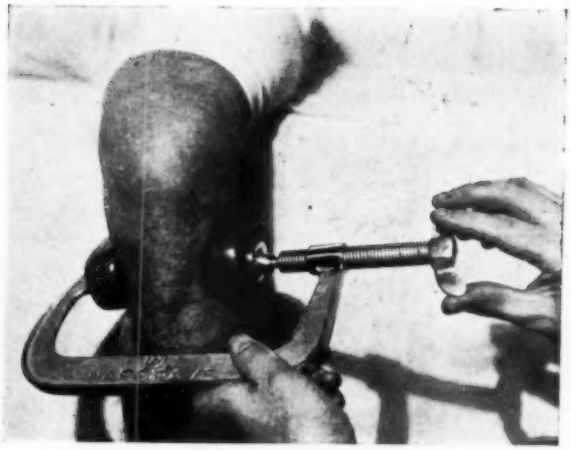

FIG. 2.

FIGS. 1 and 2. Author's new tibial redresser.

FIG. 3.

FIGS. 3–5. Case treated by old method of hand manipulation.

FIG. 4.

FIG. 5.

FIGS. 6–9. Case treated by use of author's redresser.

FIG. 6. Before reduction.

FIG. 7. After reduction, showing skin fitting plaster cast.

FIG. 8. Lateral view taken after reduction and application of skin fitting cast.

FIG. 9. After reduction and application of skin fitting cast, showing method of cutting cast and applying knee joint hinges for purpose of obtaining early motion.

FIGS. 10–12. Another case treated by new method with redresser.

FIG. 10. Before reduction with new instrument.

FIG. 11. After reduction with knee hinges in position.

FIG. 12. Two months later, showing amount of motion before any physiotherapy or massage.

wobbly joint, and excellent results. However, this is not always the case. With the use of the *redresseur* the element of the wobbly knee joint or possible limitation of motion is entirely eliminated.

In Figures 6 and 10 the amount of comminution and displacement of joint surface in two cases is evident. In the second step with the *redresseur* one may note the excellent reduction and restoration of joint surface with an excellent weight-bearing line (Figs. 7 and 11).

Following the reduction it was noticeable that the application of the cast did not change the position of the reduced fragments, indicating that the logical time to do this is a week to ten days after fracture, at which time the organized deposit about the fracture helps to hold it following reduction. Immediately a stockinette is applied from the toes to above the knee, as high on the thigh as possible. A Böhler cast, which is a skin-fitting cast with no padding, is then applied over the entire limb from the thigh to the toes. In Figures 7 and 8, the cast is applied with no padding.

In a week to ten days after reduction and application of the cast, and as soon as the pain has subsided, I applied my knee joint hinges, as shown in Figure 9. A window was removed from both the anterior and the posterior portion of the knee, semicircular in shape, above and below the knee. The patient was then instructed to manipulate the splint by flexing the joint increasingly each day, so that beginning with 5 degrees' flexion the first day, it should be increased to 10 or 15 degrees the following day, and so on until full flexion has been obtained.

I want to emphasize one thing that must be carefully observed, and that is that the patient should be instructed to extend the knee fully at night, and every night, because if this is not done there may form a bony deposit in the anterior chamber of the knee which will lock it and limit full extension.

As one will note in the *x*-rays taken following the use of this modified implement, the reduction is very gratifying as compared with the one in which I did not use this implement. The plaster casts were removed in these 2 cases at the end of eight weeks and measurements showed that even before any physiotherapy, message, or muscle education had been instituted, these patients had full extension, with 90 degrees' flexion in one case and 95 degrees' flexion in the other. Weight-bearing can be begun with a cane at the end of ten weeks and recovery is complete in five to six months.

It is fallacious to state that these patients can recover any quicker than this. There are too many conditions entering into the situation to permit earlier recovery.

One may be inclined to say that is all right for a specialist who has these implements at hand to do this type of work, but in any carpenter's shop anyone can find an instrument which is used for holding three or four planks together when they are glued, called a wood clamp, which, if heavily constructed, would be just as effective as a metal instrument.

You will find that you will not get any pressure necrosis, because it takes only four to five minutes to accomplish the reduction. You will also find that it takes a great deal of pressure to reduce this type of fracture, much more than you have any idea of, so one must not be afraid to exert a great deal of pressure in the reduction of this fracture.

I strongly advise a comparison of both knees before reduction so as to determine the shelving deformity that exists where the outer displacement is present. If the injured knee is first compared with the normal one, then it is easy to reduce the displaced fragments in proper line by comparison with the normal.

CHRONIC APPENDICITIS—DUODENAL ULCER

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TRADITION in surgery is a potent influence; fads and fancies do not endure. Progress is along the path between the old and the new. In the study of the cause and cure of disease, infection has been the major consideration. The craft of surgery has advanced so rapidly and so far that, as Moynihan says, little more can be expected of it in the future. The surgery of peptic ulcer has progressed along the lines of strict empiricism. The cause of peptic ulcer, upon which rational treatment should be based, is still a subject of controversy. In the study of this problem, infection has been the dominant theme which has been supported by two major factors. The surgical observation that duodenal ulcer nearly always has associated with it infection elsewhere in the abdomen, usually in the appendix which if not removed may cause a recurrence of symptoms and sometimes a new ulcer; and the theory of the elective localization of bacteria.

The most important advance in the etiology of peptic ulcer is the fact that the lesion with all its clinical and pathological characteristics can be produced experimentally, without the factor of infection, by a method which apparently proves that the hydrochloric acid of the stomach is the direct causative agent.

It has long been known that the function of the digestive organs is profoundly affected by nervous influences. The term "functional dyspepsia" has been out of fashion since the clinical vogue of duodenal ulcer. Perhaps we have been too hasty in casting the old aside. The newer terms like appendiceal dyspepsia and duodenal ulcer in place of hyperchlorhydria, have not explained the functional derangement of the stomach. There seems to be general satisfaction with the theory that the symptoms which are associated with ulcer are directly caused by the ulcer. However, the experience of many surgeons will attest the fallacy of this doctrine because it is not uncommon in the operating room to reveal the absence of ulcer in the presence of typical symptoms. In many such instances a diseased appendix is the responsible lesion.

Surgical leaders as Moynihan, Deaver and W. J. Mayo have repeatedly stressed the association of appendicitis and duodenal ulcer and have advocated the necessity of appendectomy in conjunction with the surgical treatment of peptic ulcer. Among the reasons for recurrence of symptoms after gastroenterostomy, Moynihan mentions failure to remove the appendix. It has naturally followed that appendicitis has been held in etiological relationship with duodenal ulcer. On the basis of clinical experience, it cannot be denied that there is some association between the two lesions. Among the many theories to explain this relationship, the most prominent is the transmission of infection along lymphatic pathways from the appendix to the cap of the duodenum.

Of course it has been assumed that infection is the cause of ulcer. Foci of infection in the mouth and in the upper respiratory tract have been considered in causal relationship with duodenal ulcer.

For obvious reasons the appendix appears more important in the theory of focal infection as applied to duodenal ulcer. The idea of a focus of infection as a cause of a distant lesion, in most instances, implies that the infection in this focus is clinically not acute and usually does not produce local symptoms. Chronic appendicitis has not been generally accepted as a clinical

entity which perhaps is due to a misunderstanding rather than to a real difference of opinion. Any surgeon of only average experience must admit that it is a common occurrence in the operating room to find an appendix which shows pathological changes characteristic of chronic inflammation, although in the history of the patient there have been no acute attacks or local symptoms. From the standpoint of pathology, chronic appendicitis must be regarded as a definite lesion in spite of any controversy regarding its status as a clinical entity.

The appendix, as an integral part of the gastrointestinal tract, when affected by inflammation, may give rise to symptoms which are manifested elsewhere in this anatomic chain, as for instance by the stomach. It is a fact that the earliest symptom in an attack of acute appendicitis usually is epigastric pain. There is clinical evidence to indicate that this initial pain is due directly to a disturbance of the pyloric region of the stomach. Nausea or vomiting is a sequential symptom. It is logical to assume that a minor degree of chronic inflammation in the appendix may produce periodic or recurrent gastric symptoms. Appendix dyspepsia is recognized by many eminent clinicians. Hurst has recently written: "It is clear, from what I have said, that in the majority of cases the symptoms of appendix dyspepsia are sufficiedtly distinctive to prevent an error in diagnosis. Sometimes, however, the symptoms of ulcer, especially of duodenal ulcer, are so closely simulated that a correct diagnosis cannot be made without further investigation." Moynihan's article published in 1910 on "Appendix Dyspepsia" is a classic. In it this master of surgical observation described the living pathology associated with many cases of chronic appendicitis. He drew attention to the irritable and spastic behavior of the pyloric region of the stomach in patients who exhibited symptoms of ulcer and in whom at operation no other lesion except a diseased appendix could be found. According to Moynihan, the symptoms of ulcer may be exhibited with great fidelity in cases where no structural lesion can be found in the stomach or duodenum. In many of these cases, the only pathological change discovered during operation is a chronic inflammation of the appendix. He believed that the gastric symptoms were due to an exaggerated action of the pylorus which could be recognized by inspection of the stomach.

In view of the recognized disturbance of the pyloric region of the stomach in many cases of chronic appendicitis with symptoms that closely simulate those of ulcer, although the absence of ulcer is proved at operation; and again the frequent, almost constant association of chronic appendicitis with duodenal ulcer—these surgical axioms have naturally led to speculation regarding the causal relationship between chronic disease of the appendix and duodenal ulcer.

The mind of the modern surgeon thinks in terms of infection. Obviously there are only two possible routes by which infection from the appendix can reach the cap of the duodenum; namely, by the lymphatic route and by the blood stream. A convenient lymphatic pathway from the appendix to the region of the duodenum has been demonstrated. Berceanu examined the cadavers of 51 newborn children and by the injection method showed that there are appendicular lymphatic glands in the duodeno-pancreatic region to which the lymph from the appendix may pass directly under normal conditions.

In a thorough experimental inquiry by Braithwaite it was shown that there is a direct lymphatic connection between the appendix and the region of the duodenum, but under normal conditions the duodenum could not be reached except by aberrant or retrograde flow. Therefore, to make possible the duodenal destination of lymph from the appendix he pictures an ascending lymphangitis and lymphadenitis which deviates the natural flow of lymph so that it easily reaches the duodenum.

Rosenow's theory of the elective localiza-

tion of bacteria utilizes the blood stream as the system of communication between the focus of infection and the lesion under discussion.

These and many other theories of the infectious etiology of duodenal ulcer are plausible enough from the standpoints of pathology and experimental surgery, but they completely ignore and utterly fail to answer the most important characteristics and questions which arise in the study and management of clinical duodenal ulcer.

One of the chief clinico-pathological features of duodenal ulcer is its chronicity with periods of remission and reactivation. Acute duodenal ulceration can be produced by any one of a variety of experimental methods; but a chronic duodenal ulcer has never been produced experimentally in an intact gastrointestinal tract. The theories of infection, thrombosis, embolism and deficient blood supply are inadequate for the explanation of chronic ulceration in the duodenum. One fact of importance stands out in all experimental work on ulcer and that is, any acute traumatic or infectious lesion in the normal stomach or duodenum undergoes prompt healing and does not recur. It has been stated that the normal tendency of chronic peptic ulcer is to recur. It would be more in keeping with the facts to state that the normal tendency of chronic peptic ulcer is to heal. Reparative processes take place in the ulcer during periods of remission of symptoms but rarely does complete healing occur. It does not seem reasonable to attribute the chronicity of peptic ulcer to some inherent factor in the ulcer such as a specific organism. The most essential factor in the whole ulcer problem is not the direct cause of ulcer but the reason for its chronicity or failure to heal. Therein lies not only the explanation of peptic ulcer but of its recurrence after symptomatic cure. In spite of the fact that typical symptoms constitute a reliable indication of the presence of duodenal ulcer, yet such symptoms often occur and recur in the absence of ulcer and the symptoms of positive ulcer may undergo remission and complete disappearance for a time although the ulcer may be partially but not completely healed. The symptoms which are indicative of ulcer are not caused by ulcer but are brought about by dysfunction of the pyloric sphincter.

There is no other lesion of the human body comparable to peptic ulcer. At times one hears attempted comparisons from the standpoints of pathology and symptomatology with varicose ulcer of the leg and anal fissure. The absurdity of such views should be readily apparent. There is only one condition which always faithfully resembles peptic ulcer in symptomatology and that is pylorospasm. In surgical experience one frequently encounters cases in which the preoperative diagnosis (fortified by all indicated laboratory tests) cannot separate the famous trinity of lesions, namely, chronic appendicitis, chronic cholecystitis and duodenal ulcer. In such cases exploration of the abdomen may reveal the three lesions, combinations of any two, or one alone. In the latter instance it is usually a diseased appendix. Even in retrospect, one must acknowledge a similarity of symptoms which is as close as the resemblance of homologous triplets. More evidence, clinical and experimental, could be submitted in support of the theory that the symptoms of uncomplicated duodenal ulcer are the direct result of pylorospasm. Likewise the gastric symptoms associated with appendiceal disease come about through reflex dysfunction of the pylorus. In previous publications have been stated the reasons for my belief that pylorospasm is an important part of the disturbance of gastric function which brings about the origin and maintenance of duodenal ulcer. This view is a reversal of the one generally accepted.

The surgical treatment of duodenal ulcer in accord with the foregoing theory of its origin should be directed primarily to the correction of pyloric dysfunction. This can be done safely and easily by removing the anterior half of the pyloric sphincter.

The technical details of the procedure have been described elsewhere. The clinical results which have been published conjointly by the late Dr. John B. Deaver and myself are encouraging enough to warrant continued use of this operation.

A normal appendix in adult life is an unusual finding. Some of the marks of abnormality are expressed in the pathology of chronic appendicitis and include round cell infiltration, lymph-follicle hyperplasia, formation of fibrous and hyaline tissue and mucosal changes. Gross structural changes have to do with varying degrees of stenosis of the lumen and the effects of obstruction; with adhesion and fixation of the appendix to nearby structures; and with fixed misplacements of the appendix such as under the terminal ileum, behind or to the outer side of the cecum. It is generally admitted that the appendix may constitute a focus of chronic infection. Are the pathological changes which affect the appendix without important bearing on the function of the gastrointestinal tract? A consideration of clinical experience and authoritative opinion discloses evidence which implicates disease of the appendix in dysfunction of the ileocecal sphincter and especially in spasm of the pylorus.

The pyloric sphincter is extremely sensitive to peritoneal irritation, a fact which can be explained only as a reflex phenomenon. The pyloric reflex is manifested as spasm which varies in degree from achalasia (failure to relax) to hypertrophy of the pyloric sphincter.

Dysfunction of the pyloric sphincter is one of the two essential factors in the causation and chronicity of duodenal ulcer and in the development of postoperative ulcer. The other factor is hyperacidity. Neither factor alone can produce ulcer. Pyloric stenosis or obstruction from any cause does not increase the concentration of hydrochloric acid in the stomach. According to the dictum of Pavlov, the stomach under normal influence secretes the exact quantity of hydrochloric acid (no more, no less) which is needed for the optimal concentration in the gastric phase of digestion. The so-called appetite or psychic secretion is the most potent factor in gastric digestion. It is controlled by influences reaching the stomach through the vagus nerves. The other and less important part of gastric secretion arises as the result of the presence of food in the stomach. Experiments have shown that this latter secretion may be adequate after the vagus nerves have been cut. The concentration of acid produced by the stomach under normal influences is fixed at about 0.5 per cent. Partial neutralization to a concentration that is optimal for digestion is brought about by reason of ingested material and probably by an inherent regulatory power of the stomach. The call for more acid comes when the optimal concentration falls and there is yet more digestive work to be done. The acidity of the material ejected from the stomach must not be too high, otherwise there will be injury to the duodenal mucosa.

Morton has shown that under normal conditions the material on the duodenal side of the pyloric sphincter is always alkaline or only faintly acid, but in the patient with duodenal ulcer it is distinctly acid. The experimental work of Mann on the production of peptic ulcer clearly demonstrates that acid is the factor of major importance.

Hypersecretion and hyperchlorhydria, by which is meant the production of acid in excess of digestive needs, is a common occurrence and it might be asked why duodenal ulcer does not develop in all or most such instances if our theory is tenable.

The explanation, we believe, lies in the regulatory mechanism of duodenal regurgitation. Under normal conditions there is probably very little regurgitation of duodenal contents into the stomach. When for any reason gastric acidity becomes excessive and beyond the control of the stomach then the safety mechanism of duodenal regurgitation serves as a neutralizing agent. The excess acid is not neutralized in the duodenum but this must take

place before it leaves the stomach. Elman and Olch have shown that duodenal regurgitation is deficient or absent in patients who have duodenal ulcer.

Anatomic studies have shown that the pyloric sphincter is a true sphincter. In it are muscle fibers so arranged, as in the iris of the eye, that they can produce actual dilatation. The larger group of fibers, circularly arranged and under different nervous control, serve to contract the sphincter. The sphincter at rest is a balanced mechanism. Failure to relax or slight spasm offers an impediment to the outflow of gastric contents; but more particularly it totally prevents in time of need the regurgitation of duodenal contents into the stomach. The reserve peristaltic force of the stomach can and does overcome this functional obstruction but the weak reverse peristalsis of the duodenum is unable to do so. These are facts confirmed by *x*-ray and other studies. Given the two conditions, namely, persistent gastric hyperacidity and dysfunction of the pyloric sphincter represented by achalasia or spasm, the result is forcible ejection of hyperacid material which strikes the duodenal mucosa at a more or less fixed point that sooner or later forms an ulcer. The role of chronic appendicitis in the production of duodenal ulcer has to do with its influence on reflex pylorospasm. Chronic appendicitis is only one, but probably the most important, of the abdominal lesions which have a reflex effect on the pyloric sphincter. Disease of the gall bladder may act in the same way. There are other causes of pylorospasm which may originate outside the abdomen.

In case of a possible misunderstanding, it should be stated that appendectomy is not advocated as a cure or preventative for duodenal ulcer. It is possible, although beyond means of proof, that in some cases appendectomy has prevented the development of duodenal ulcer. From our surgical experience we have demonstrated that when the pyloric sphincter is rendered incapable of obstructing duodenal regurgitation, a duodenal ulcer is clinically cured. The operative procedure, as described in previous publications, is merely the submucous removal of the anterior half of the pyloric sphincter.

CONCLUSIONS

1. Chronic disease of the appendix and the local changes remaining after an acute inflammation of the appendix constitute a focus of persistent peritoneal irritation.

2. These changes in and about the appendix may be a cause of persistent reflex pylorospasm. The changes in the pyloric region of the stomach, especially dysfunction of the sphincter, are not directly due to infection but are reflex phenomena from peritoneal irritation about the appendix.

3. Spasm of the pyloric sphincter is one of the two essential factors in the development of duodenal ulcer. The other factor is hyperchlorhydria.

4. The etiologic bearing of chronic appendicitis on duodenal ulcer is through its influence on the function of the pyloric sphincter.

5. The logical and practical surgical treatment of duodenal ulcer as well as the prevention of postoperative ulcer is the correction of pyloric dysfunction by removal of the anterior half of the pyloric sphincter and appendectomy.

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[For Remainder of References see p. 241.]

GASTROJEJUNAL ULCER IN CHILDHOOD

REPORT OF A CASE

J. E. STRODE, M.D., F.A.C.S.

HONOLULU, T. H.

THE occurrence of definitely proved chronic peptic ulcer in childhood, judging from the literature, is a rarity. Proctor[1] of the Mayo Clinic in 8260 peptic ulcers observed between 1906 and 1925 found only 2 cases, one gastric and one duodenal. In a search of the literature he found only 19 definitely proved chronic ulcers in patients under the age of fourteen. Of this number 10 were subjected to operation, and no mention is made of their subsequently developing a marginal ulcer. I have been unable to find mention of this complication, and therefore believe the following case report to be of sufficient interest to justify recording.

The patient, a Chinese male of ten, came under our care in February, 1931, complaining of severe epigastric pain, nausea, vomiting and loss of weight. The symptoms were of three years' duration, beginning as mild epigastric pain lasting a few days with a remission of two to four months, recurrence, increasing severity, and longer duration. The epigastric pain had never borne any relation to eating. Two years after the onset of symptoms the patient was treated medically for four to five months, being on a diet consisting principally of milk, cream and crackers. Marked improvement was noted for a time and then symptoms recurred. An *x*-ray examination at this time showed deformity of the cap with marked retention. On examination the only positive findings were emaciation, anemia and moderate epigastric tenderness. The red blood count was 4,900,000, hemoglobin 56 per cent. Urine was negative. It was evident that the patient was suffering from starvation, the result of pyloric obstruction, and operative interference seemed indicated. His dehydration was corrected by intravenous saline and glucose and immediately preceding operation he was given 300 c.c. of citrated blood.

An ulcer, on the point of perforating, was found on the anterior superior portion of the duodenum just beyond the pylorus. There was marked edema surrounding the ulcer and periduodenitis. The ulcer was sewn over and covered with omentum. A posterior, no loop, gastroenterostomy at the most dependent portion of the stomach was done three rows of fine chromic catgut being used. The patient made an uneventful postoperative recovery and left the hospital at the end of a week. He weighed 52 pounds, and three months later 57 pounds, and was feeling fine.

He was next seen in October, 1931, (seven months postoperative) because of epigastric pain and vomiting. Blood examination at this time showed 3,300,000 red cells with achroma, poikilocytosis and anisocytosis, hemoglobin 30 per cent, white blood count 9850, and stool positive for blood on a meat-free diet. Though is was strongly urged that the patient enter the hospital for study this was not done for two months. Meanwhile the epigastric pain had become much more severe, the patient more anemic and emaciated from vomiting and fear of eating because of provoking pain. There was tenderness in the epigastrium especially to the left of the umbilicus. Blood was still present in the stools, hemoglobin 40 per cent, and red cells 1,860,000. Fluoroscopic and *x*-ray study of the stomach showed no barium passing through the pylorus; a small amount of residue at six hours, a gastroenterostomy opening that appeared small and irregular with a collection of barium to the left of the stoma suggesting an ulcer crater.

Three blood transfusions of 300 to 400 c.c., each twenty-four hours were given with marked improvement in the patient's appearance, and rise of hemoglobin to 50 per cent and red blood cells to 3,520,000. At operation a large ulcer was found in the jejunum just to the left of the stoma penetrating into and prevented from perforating by the transverse

[1] Proctor, O. S. Chronic peptic ulcers in children. *Surg. Gynec. Obst.*, 41: 63, 1925.

mesocolon. The sight of the duodenal ulcer was indurated and surrounded by adhesions, not edematous, and appeared healed or at least

Fig. 1. Gastrojejunal ulcer. Arrow points to ulcer crater.

quiescent.

A subtotal gastric resection including the limbs of the anastomosis was done, repair consisting of end-to-end anastomosis of the jejunum and Moynihan type II gastrojejunostomy. The patient stood the operation well, was given 300 c.c. of citrated blood before leaving the table, and discharged from the hospital one week later.

He was last seen on July 23, 1932 at which time he weighed 68 pounds and was free of gastric symptoms.

While in previously reported cases of peptic ulcers in children hyperchlorhydria has usually been absent it would have been of much interest to know the acid content of the stomach at the various stages in this case because of the development of the jejunal ulcer. A high HCl following simple gastroenterostomy some believe to be responsible for the developing of marginal ulcers, and this is an argument used by advocates of the more radical

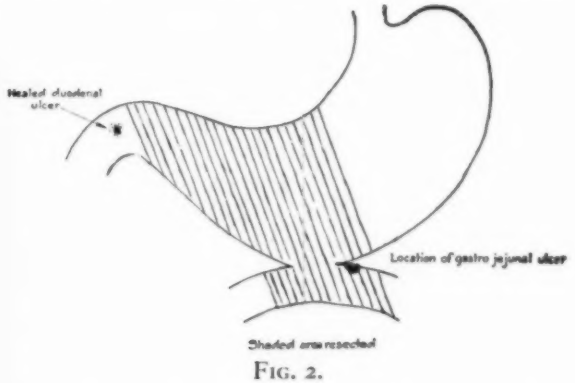

Fig. 2.

operation of subtotal gastric resection. Unwillingness of the parents to cooperate made scientific curiosity secondary to other measures of more practical value.

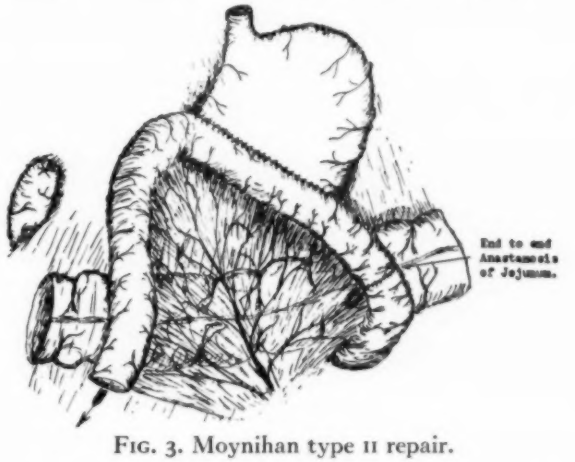

Fig. 3. Moynihan type II repair.

While the number of proved peptic ulcers in childhood has been small it is quite likely that many of them go unrecognized as careful history taking has in adults with ulcers not infrequently unearthed a train of epigastric symptoms dating back to childhood.

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RENOCOLIC FISTULAE*

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RENOCOLIC fistula is mentioned by Hippocrates[1] as a rupture of a renal abscess into the intestines. Rufus,[2] Avicenna,[3] and Fernel[4] also record observations, but possibly the most complete study was made by Rayer.[5] In his thorough fashion he includes renocolic fistula in his "Traité des maladies des reins et des alterations de la secretion urinaire," devoting considerable space to a consideration of its various ramifications. Rayer evidently believed that prior to the establishment of a renocolic fistula there was a long period of chronic inflammation of the kidney, frequently tuberculous in character but not necessarily always so. The kidney became distended with pus associated with severe colic, a lumbar tumor, and some cutaneous rubor. Throughout this period the adjacent colon became attached to the inflamed kidney by fibrous adhesions and developed in turn a chronic inflammation and ulceration. Eventually a hectic fever made its appearance, followed not infrequently by diarrhea of pus and urine with the sudden obliteration of the lumbar mass and a relief of the symptoms. Even at the present time, we can hardly offer any more satisfactory history of the disease, as will be seen from the following study.

The outstanding findings of the various case reports as given by Rayer and others are renal colic, usually intermittent in character, a tumor in the flank which usually has a history of suddenly disappearing with marked relief of symptoms, and the finding of pus and urine in the stools. However, as Rayer observed, the size and tensity of the tumor mass depend not a little on the extent to which the pus is able to evacuate itself through the fistulous tract.

A recent excellent paper summarizing all the reported cases to date, giving symptoms and autopsy or other findings, is that written by Mertz[6] in 1931.

Rayer does not believe examples of renocolic fistula to be rare, and cites cases from Fatoni,[7] Duverney,[8] Baillie,[9] Weigel,[10] Wys,[11] Lassus,[12] Pierre Frank,[13] and others, but we seldom see one today. It would seem that at least part of the reason for the diminution in the number reported in the recent literature is due to the prodromes associated with the development of this condition. Few patients would tolerate the renal colic associated with the tumor, and few surgeons would hesitate to drain a pyonephrosis or perinephric abscess before a fistula establishes itself. Nevertheless, that the condition still occurs is shown in the case reported from this clinic as well as the 2 cases reported by Mertz.[6] In Mertz's article he mentions 10 other instances which have been reported since Rayer's publication in 1841. In addition, we must mention Chazet's[14] recorded in his Thèse de Paris for 1900 and also Blanco's[15] in 1930. He records the accidental discovery of a renocecal fistula while making a right urogram, at which time the patient developed intestinal colic and uncontrollable defecation. The radiogram showed the opaque medium passing from the kidney into the cecum.

CASE REPORT

The following is the report of a case recently seen and operated upon with the details of the clinical and operative findings.

The patient, J. F. No. A4277, a woman thirty-nine years of age, was first seen in the New Haven Dispensary on October 16, 1931. She complained of pain in the right side which had

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been present off and on for the past seventeen years but which had been considerably worse during the past two years.

Family and personal history were entirely irrelevant, as apart from the present illness the patient had been entirely well and free from any other symptoms. She was married and had three living children.

Present Illness. For the past seventeen years the patient had had repeated attacks, at infrequent intervals, of a sharp nagging pain in the "region of the gall bladder" and the back on the right side. The attacks lasted for two to three weeks at a time and were made worse by "food that is hard to digest" and were frequently associated with nausea and vomiting. After vomiting the pain disappeared.

Two years ago the patient was seized with an attack so severe that she had to quit all work. The pain was localized to a point just under the ribs and in the right side of the back. It was associated with the vomiting of green fluid material. Four months later a still more severe attack occurred which was so agonizing that the patient broke out in a cold sweat. It was again localized to the same area and was associated with vomiting. For the few weeks prior to coming to the dispensary, the attacks of pain and vomiting, although not so marked, were recurring as often as twice a week. Recently they had been relieved by hot water bottles.

Although the patient had had no urinary symptoms in the form of burning, smarting, frequency, urgency or pyuria, she noticed that after an attack of pain the urine would become cloudy and then gradually become clear again.

Physical Examination. The patient, who was thirty-nine years of age, was a well-developed, well-nourished woman, although she was perhaps somewhat anemic. Examination of head and neck, heart and lungs revealed no gross abnormalities.

The abdomen was flat and soft and the abdominal muscles had poor tone. No masses were palpable, the kidneys were not felt but definite tenderness was present just below the costal margin in the right epigastrium; there was also some tenderness in the right costovertebral angle. No other areas of tenderness were present. There was no muscle spasm and no rigidity. The percussion note was tympanitic throughout.

Pelvic and rectal examinations revealed only a moderate degree of cystocele and rectocele. All reflexes were active and equal.

On October 16 the patient had *x*-rays of the gall bladder and later a barium clysma. These revealed a normally functioning gall bladder and a normal large bowel. During this examination a shadow suggestive of a right renal calculus was noted so the patient was referred for cystoscopic and urographic examinations.

Cystoscopic Examination November 2, 1931. A size 24 F. cystoscope was readily passed into the bladder. The bladder capacity was 425 c.c. The bladder mucosa was diffusely injected but no ulceration, tumor, stone or diverticulum was seen. The ureteral orifices appeared normal and seemed to function rhythmically and at regular intervals. Size 5 F. ureteral catheters were readily passed up each ureter to its kidney pelvis without obstruction.

Urine Examination. The urine from the bladder was grossly hazy, amber in color, specific gravity 1.016. There was a very faint trace of albumin present; no sugar; a moderate number of pus cells and many bacilli which on culture were shown to be B. coli.

The right kidney urine was somewhat mucoid and reddish brown in color. Microscopically it contained mucus, pus cells and bacilli which on culture were shown to be B. coli.

The left kidney urine was clear and microscopically contained no pus cells nor organisms. Cultures also were negative.

Intravenous 'phthalein appeared in five minutes from the left kidney, and during a thirty-minute period of collection 23 per cent of the dye was excreted from this kidney. Only a faint trace of the dye was present in the urine from the right kidney at the end of the half-hour period of collection. The patient's bladder was catheterized at this time but there was no urine present, which would indicate that the divided 'phthalein determination might be considered accurate.

X-rays. A radiogram of the urinary tract revealed an opaque shadow overlying the lower pole of the right kidney, suggestive of a renal calculus. A urogram of the right tract showed a marked irregularity of the shadow cast by the renal pelvis and calyces, and an apparent extension of the opaque medium into the ascending and transverse colon through what appeared to be a small fistulous tract connecting the inferior calyx with the ascending colon (Fig. 1). A residue film taken ten minutes later (Fig. 2) showed the major portion of the opaque medium to have passed from the kidney

pelvis and calyces into the large bowel. The urogram of the left tract revealed no abnormalities.

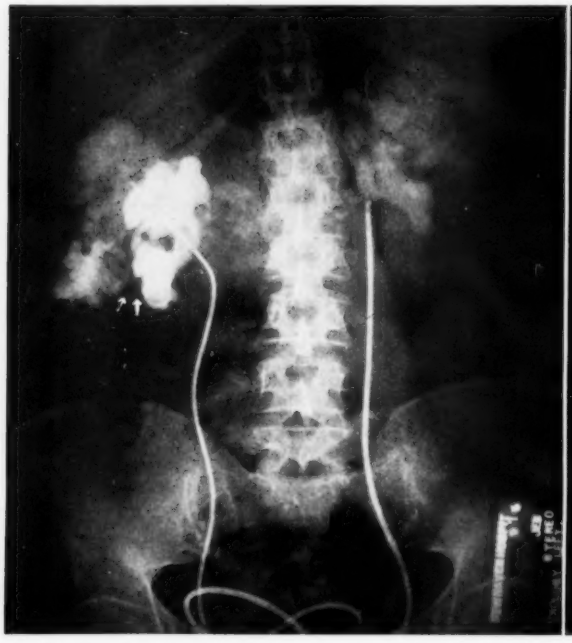

Fig. 1. Stereoradiogram of urinary tracts after injection of 10 c.c. of opaque medium into each renal pelvis through size 5 F. ureteral catheter. Note on right side (1) marked irregularity of shadow cast by renal pelvis and calyces; (2) slightly negative shadow in lower calyx due to renal calculus which was readily seen before injection of opaque medium; (3) fistulous tract (indicated by arrows) extending from lower calyx to ascending colon; and (4) opaque medium in ascending and transverse colon.

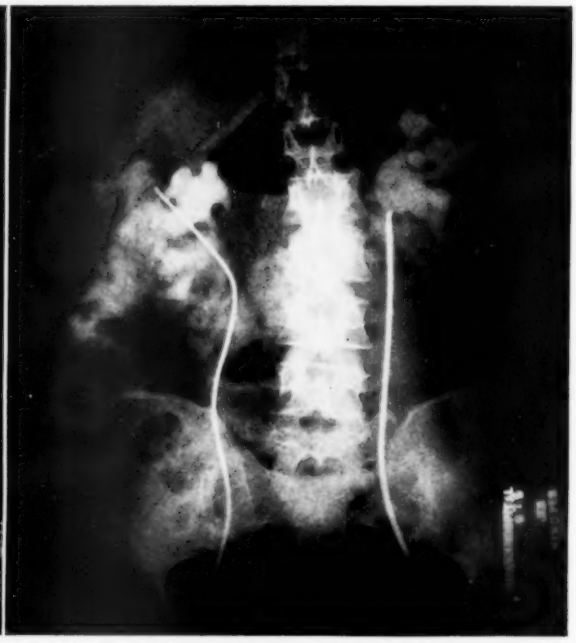

Fig. 2. Radiogram of urinary tracts made ten minutes after that shown in Figure 1 without further injection of opaque medium. Note apparent emptying of contents of right renal pelvis into large intestine.

The patient was admitted to the New Haven Hospital on November 4, 1931 for further study and treatment.

Repeated cystoscopic examination revealed findings similar to the foregoing. Repeated fluoroscopic and radiographic examinations with the foregoing findings in mind revealed no primary pathologic changes in the large bowel, and the fistulous tract connecting it with the right kidney which was seen in the urogram was not demonstrable.

Intravenous 'phthalein revealed a function of 40 per cent the first hour and 15 per cent the second hour. Blood non-protein nitrogen 31 mg. per liter. Blood count: red blood corpuscles 3,200,000; hemoglobin 50 per cent; white blood corpuscles 7600. Blood Kahn test negative.

Operation November 12, 1931. Exploratory laparotomy; closure of fistula between right kidney and ascending colon; nephrostomy.

Anesthetic. Avertin supplemented by ethylene.

Procedure. To obtain a satisfactory exposure the combination of a right rectus incision and a second incision at right angles to this and running parallel to the lower costal margin was used.

When the peritoneal cavity was opened the large bowel presented. The entire ascending and transverse colon was carefully examined and no evidence of tumor or induration could be found. The upper half of the ascending colon and the hepatic flexure were densely adherent to the posterior abdominal wall and presumably to the right kidney. The stomach and gall bladder were normal.

The posterior peritoneum was now incised preliminary to freeing the kidney which was found to be bound down everywhere by very dense adhesions, evidently from a long-standing perinephritis or a healed perinephric abscess. The dissection, which revealed a large scarred kidney with very little functional renal substance remaining, had to be done almost entirely by sharp dissection. Anteriorly the ascending colon was densely adherent over the lower half of the kidney. Between these two structures a small fistulous tract which was not more than 3 mm. in diameter was divided.

The opening into the bowel was readily closed by a purse-string suture of fine black silk reinforced by two mattress sutures of fine black silk as there was very little induration or infiltration of the bowel at this point. The opening into the kidney overlay the calculus and was in the midline of the kidney anteriorly at the junction of its middle and lower thirds.

The dissection being carried farther medially, the large vessels were exposed. A short renal vein and many dense adhesions bound the medial surface of the kidney to the vena cava.

The patient's general condition did not warrant doing any more at that time so the peritoneum was closed, the opening into the kidney enlarged and the wound closed and drained.

The patient recovered satisfactorily from the operation but on November 22, 1931 she developed pneumonia from which she unfortunately did not recover. Permission for an autopsy was not obtained.

DISCUSSION

It is interesting to observe that of the 26 cases reported in the literature that give some pathological details, 8 were associated with renal calculus, only 2 were associated with tuberculosis, while 13, or one-half, are described as being "pyonephrotic" while 12 had either a chronic or an acute suppurative perinephritis.

What is still more interesting is to note that in no instance was there any associated primary pathological change in the large bowel.

In interpreting these findings let us review the anatomical relations of the anterior surface of the kidneys and also their relation to the reflection of the peritoneum from the posterior abdominal wall.

The large bowel comes directly in contact with the anterior surface of the lower third of each kidney although this contact is more extensive on the right side than on the left, as can be seen in the accompanying diagrams (Fig. 3A and B) which shows the line of reflection of the peritoneum from the posterior abdominal wall.

At the hilus of the right kidney the duodenum comes in direct contact with its anteromedial surface. The tail of the pancreas takes the place of the duodenum on the corresponding surface of the left kidney.

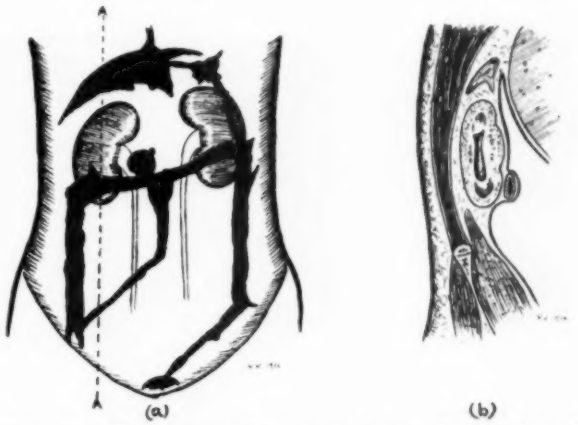

Fig. 3. A. Diagrammatic representation of reflection of peritoneum from posterior abdominal wall (after Delepine). Kidneys and upper ureters are drawn in to show relation of peritoneal reflection (represented in solid black) to them. Note how lower third of each kidney is uncovered by peritoneum.

B. Peritoneum covering posterior abdominal wall at location indicated by AA in Figure A. (After Gerota.) Note how peritoneum leaves surface of kidney to cover colon and also intimate relation of large bowel to lower third of anterior surface of kidney.

Superiorly the adrenal comes in direct contact with the kidneys without the intervention of the peritoneum.

Bearing these relations in mind it will readily be seen that any perirenal inflammation or suppuration will cause the relationship between the kidney and large bowel to become still more intimate, and later, as the result of pressure necrosis, ulceration may occur into the bowel forming a fistula.

If renal infection, with or without the aid of a calculus, causes sufficient erosion of the kidney substance to allow the kidney to rupture, then, in ordinary circumstances, a true perinephric abscess develops. This is to be distinguished from a perinephritic abscess which is in reality an acute suppurative process in the perinephrium, due, as a rule, to the Staphylococcus aureus, and does not necessarily bear any relation to disease of the kidney itself. The abscess will dissect between the perinephrium,

which it may later involve, and the kidney but will be held for a time within Gerota's fascia, only later to rupture through the layers of this fascia to appear inferiorly in the groin, or if it ruptures superiorly it may make itself apparent as a subdiaphragmatic abscess or later as a renobronchial fistula.

For rupture into the large bowel or duodenum, therefore, we must first have either a healed perinephric abscess or a healed or long-standing perinephritis so that the kidney and bowel are densely adherent. It then becomes easier for a small renal abscess overlying an area covered by bowel to ulcerate into the bowel than to break through the dense perirenal adhesions.

If these facts are true, then by far the majority of fistulae between the kidney and the bowel must pass through the kidney itself for the reason that we presuppose a pyelonephritis with ulceration. This too is borne out by the literature. Except for Watson and Cunningham's case[16] and that of Eugene Fuller,[17] both of which are fistulae between the kidney pelvis and the bowel, all the renocolic, as well as the only two renoduodenal fistulae recorded, are described as being between the kidney itself and the bowel.

In the case here reported the patient had been ill for seventeen years. She had had recurring attacks of pain in her right side which were subsequently associated with nausea and vomiting. These are all symptoms that can readily pertain to a renal lesion. For the past two years she had had attacks of what may readily be interpreted as severe renal colic. Each symptom in turn increased in its intensity, severity and frequency until her admission to the hospital. We also note that the patient's urine was "cloudy" after an attack of pain and gradually again became clear until the next attack.

Knowing these facts, we may infer that there had been a chronic renal infection for seventeen years with many acute exacerbations probably accompanied by a certain amount of acute and chronic perinephritis. Then two years ago a renal calculus in the lower calyx probably developed, obstructing it temporarily and forming a localized pyonephrosis. The symptoms were correspondingly aggravated until a shift in the position of the calculus gave drainage, resulting in the relief of symptoms and the appearance of a cloudy urine. During one of these episodes the pyonephrosis must have eroded through the kidney cortex into the bowel which during the previous fifteen years had become densely adherent to the kidney. At operation these findings were confirmed.

SUMMARY

Renocolic fistulae are always secondary to long-standing suppurative lesions of the kidney which as a rule are associated with either a chronic perinephritis or a perinephric abscess.

In a third of the reported cases renal calculus was associated with this lesion, while in only 2 of the 34 was the primary lesion tuberculous in origin.

In no instance was there any primary pathological process in the large bowel.

In only 2 instances did the report claim that the fistula was between the renal pelvis and bowel. In all others the fistula was from a calyx, passing through the kidney substance into the bowel.

A study of the anatomical relations of the reflection of the peritoneum from the posterior abdominal wall will give some idea as to why and where renocolic fistulae occur.

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[For Remainder of References see p. 296.]

SOME STUDIES WITH THE

MAXIMUM UREA CLEARANCE*

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INTRODUCTION

FROM time to time much interest has been manifested in the various kidney function tests and considerable research has been done in an effort to devise an accurate means of measuring the functioning ability of the kidneys. Due to the difficulty in controlling a number of variable factors it has been hard to place a correct interpretation on these tests. First one and then another has held its place as the most accurate, only to be replaced by a third and more recent.

One of the main functions of the kidney is the excretion of urea. For this reason a study of this substance both as a constituent of the blood and as an excretory product has constituted one of the most important chapters in the development of renal function tests. For a complete history of the development of urea excretion estimates of renal function I refer the reader to an article by Moller, McIntosh and Van Slyke.[1]

The blood urea has been recognized for a long time as a valuable means of diagnosing kidney damage. With ordinary urine volumes the blood urea is dependent mainly on the protein catabolism and the functioning renal tissue.

According to Moller, McIntosh and Van Slyke,[1] Ambard and Weill "were the first to include both urea output and urine volume in attempting quantitatively to relate urea excretion to blood content." Again according to Moller, McIntosh and Van Slyke the formula of Ambard and Weill simplified reads:

$$K = \frac{B}{\sqrt{D}\sqrt{U}}$$

and is only accurate when the urine volume is normal or below the augmentation limit of approximately 2 c.c. per minute. In the formula of Ambard and Weill, B and U represent concentrations of urea in blood and urine respectively, and D is the rate of urea excretion or $U \times V$, V representing the urine volume.

Taylor, Drury and Addis,[2] working on rabbits, attempted to demonstrate that the ratio

$$\frac{\text{Urea in 1 hour's urine}}{\text{Urea in 100 c.c. blood}}$$

varied in direct proportion to the weight of normal renal tissue. This ratio was believed to be independent of urine volume, but the standard conditions recommended for its determination would probably keep the output above the augmentation limit. This ratio is very similar to the maximum clearance of Moller, McIntosh and Van Slyke.

Austin, Stillman, and Van Slyke[3] in 1921 showed that the urea excretion ratio of Addis held only when the output was above a certain amount, known as the augmentation limit, which varied in individuals but averaged about 2 c.c. a minute. These authors also demonstrated that when the volume of urine dropped below the augmentation limit the excretion rate of urea varied approximately in proportion to the square root of the volume of urine. Adopting as a standard urine volume 1 c.c. a minute, because this is the approximate average normal urine volume and because it simplifies calculation by representing unity, Moller, McIntosh and Van Slyke devised a formula for measuring the standard urea excretion from any volume of

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urine under the augmentation limit. The standard clearance then measures the amount of blood cleared of urea per minute

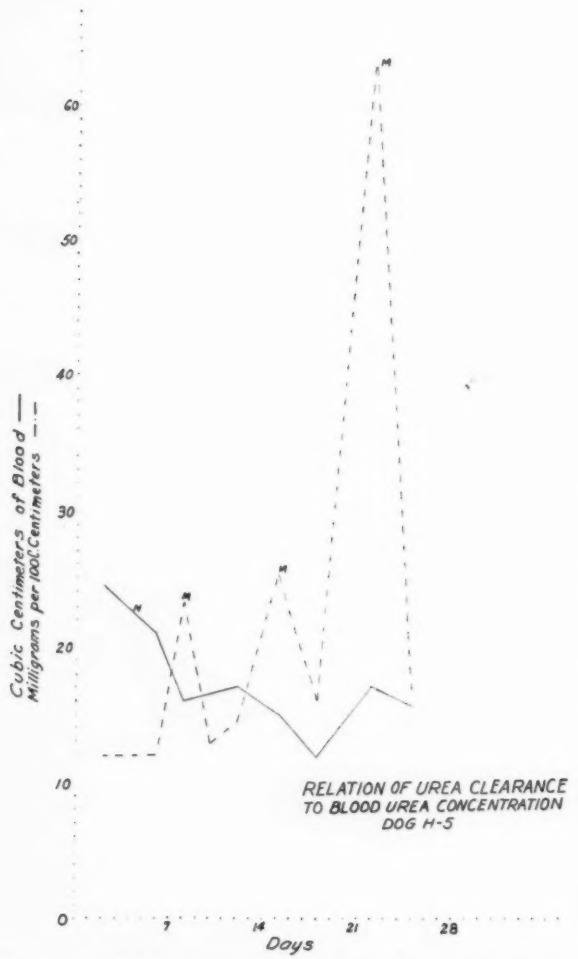

Fig. 1. Solid line, maximum urea clearance; broken line, blood urea concentration; N, nephrectomy; M, Monday.

This is a fairly typical result. The postnephrectomy urea clearance is reduced to 70 per cent of the prenephrectomy value, the renal weight to 44 per cent. The increase in the blood urea concentration ocurring on Monday, two days after a large meal of meat is well demonstrated in this figure.

when the urine volume is 1 c.c. per minute and averages for a normal adult about 54 c.c.

When the output of urine is greater than the augmentation limit the kidney is at its best and is excreting the maximum amount of urea, this maximum amount depending upon the urea concentration in the blood, or "expressed in other words, with abundant urine the urea excretion per minute equals the urea contained in a constant volume of blood. This volume of blood in a normal adult is about seventy-five cubic centimeters" (Moller, et al[1]). Thus as the concentration of the urea in the blood increases the amount of urea excreted increases, but the amount of blood cleared of urea remains the same. This maximum amount of blood cleared of urea is called the maximum clearance. Moller, McIntosh and Van Slyke are responsible for this term and have devised the following formula for measuring the maximum clearance:

$$Cm = \frac{UV}{B}$$

where Cm is the maximum clearance, U the urea concentration in the urine, V the volume of urine measured in cubic centimeters per minute and B the urea concentration of the blood.

The maximum and standard clearance values are believed to be fairly accurate measurements of the amount of functioning kidney tissue plus the volume of blood flowing through the kidneys. These last two factors, however, cannot be determined with mathematical exactness, because there are certain other minor conditions which affect the urea excretion. There is a ± 6 per cent variation in the maximum clearance and a ± 10 per cent variation in the standard clearance according to MacKay.[4] This author found a rather constant variation in the standard clearance throughout the day. He found that eating caused no definite effect, but in one instance exercise caused a marked drop in the standard clearance. He referred to a similar drop in the maximum clearance reported by Addis.[5] MacKay also reports that the larger the value of the standard clearance the greater is the variability of this figure.

Moller, McIntosh and Van Slyke[6] have shown that the "loss of renal function may exceed 60 per cent before the blood urea content rises above the highest level observed in normal subjects."

Addis, Meyers and Oliver[7] were of the opinion that unilateral nephrectomy "has no appreciable effect on such measures as are customarily regarded as being indices of renal functional capacity." After reviewing the literature they came to the conclusion that there was a general agreement "that no obvious functional defect is necessarily produced by removing one kidney." By using the ratio

$$\frac{\text{urea in one hour's urine}}{\text{urea in 100 c.c. blood}}$$

and putting the kidney under a strain by the administration of large amounts of urea, Addis, Meyers and Oliver[7] were able to get an approximate measure of the structural change after unilateral nephrectomy.

METHODS

The object of this experiment was to investigate the value of the maximum urea clearance determination as a test of renal function. The procedure was first approached by establishing average units of function per gram of renal tissue, kilogram of body weight and liter of body volume. These average units were then compared with the individual determinations and percentages of variability estimated. The value of this test was further studied by comparing the reduction in renal weight to the reduction in renal function following nephrectomy. The maximum urea clearance was finally compared to the blood urea concentration over a period of three weeks in order to determine any early compensatory hypertrophy effects and to correlate the relative merits of the two determinations as estimations of kidney function.

Adult female dogs were chosen for several reasons. Catheterization could be done much more easily than in the male, much larger catheters could be used, thereby insuring a quicker emptying of the bladder, hence a more accurate timing of the experiment. The bladder could also be more completely emptied with a larger catheter.

The urine volume of the dog was found to be very small, varying from 1 to 10 c.c. per hour. The experiments were made during the months of July and August

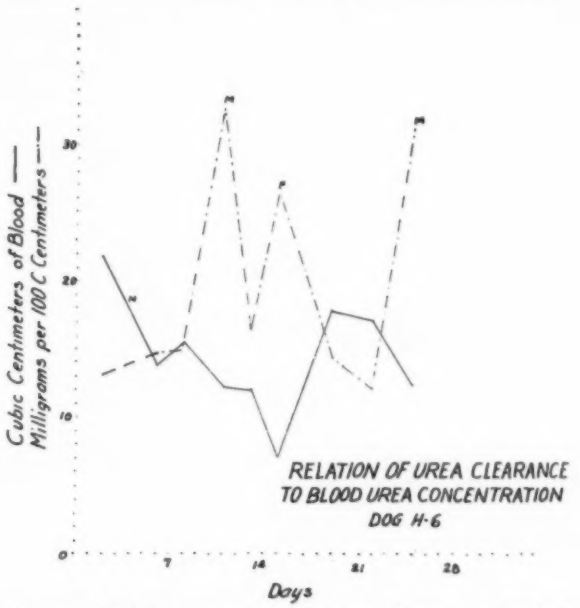

Fig. 2. Solid line, maximum urea clearance; broken line, blood urea concentration; N, nephrectomy; M, Monday; F, Friday.

The urea clearance value was reduced to 63 per cent and the renal weight to 60 per cent in this animal following nephrectomy. The relative merits of the urea clearance determination and the blood urea concentration as indices of kidney function are well demonstrated in this figure. After the initial fall the urea clearance remains below the prenephrectomy normal reading. The first observation after nephrectomy finds the blood urea concentration only slightly above the prenephrectomy value. In subsequent observations the blood urea concentration is either above or below the prenephrectomy value.

when the atmospheric temperature ranged around 85°F. The dogs panted very freely and this probably was the principal factor responsible for the low urine volume. With an output of this size it was obvious that the standard urea clearance would have a very high percentage of error. For this reason its determination did not seem worthwhile.

The urine volume could not be sufficiently increased for maximum clearance determinations when water was given only by the stomach, regardless of the quantity ingested. When large quantities of water were administered through the stomach tube the maximum urine volume obtainable varied from 0.75 to 2 c.c. a

minute. In order to get the urine volume consistently above the augmentation limit of about 2 c.c. a minute it was necessary

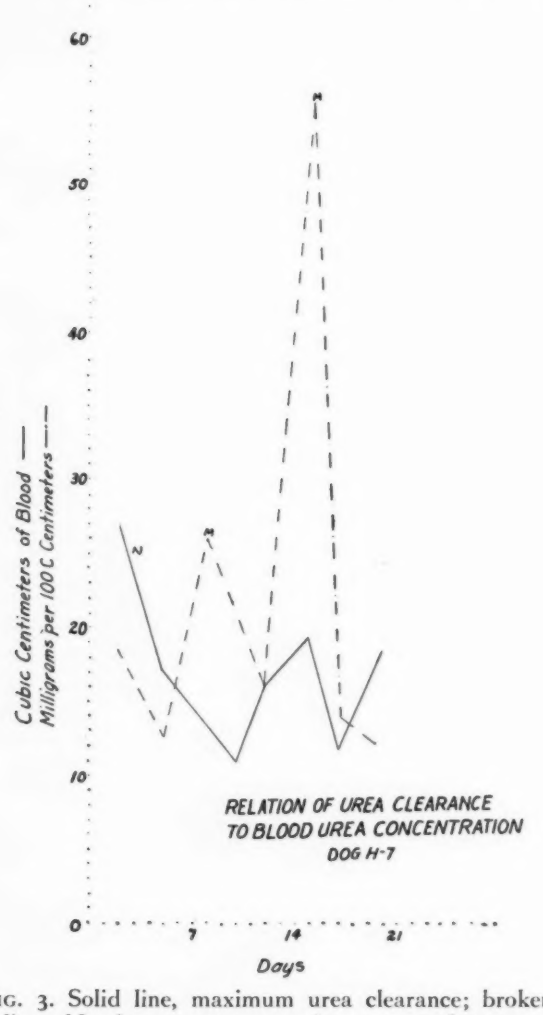

Fig. 3. Solid line, maximum urea clearance; broken line, blood urea concentration; N, nephrectomy; M, Monday.

After nephrectomy the renal function and renal weight were reduced to 50.9 and 53 per cent of the prenephrectomy values. The marked variation in the blood urea concentration as contrasted to the urea clearance is strikingly demonstrated.

to give infusions of normal saline in conjunction with the administration of water by the stomach tube. Infusions alone would not suffice unless the quantity of normal saline solution used was enormous, amounting to around 500 c.c. It was also found that unless the dogs were kept cool they would markedly diminish their output by panting.

The routine was as follows: The dogs were given water by the stomach tube and about forty-five minutes later infusions of 150 to 200 c.c. of normal saline solution, depending on the size of the dog. The bladder was catheterized immediately before the infusion and fifteen minutes after the infusion was stopped. Catheterization was done before the infusion in order to empty the bladder of all of the concentrated urine excreted previous to the test. Catheterization fifteen minutes after the infusion was done for two reasons, first to wash out the bladder with urine of approximately the same concentration as the urine on which the urea determination was to be made, and second in order to estimate the approximate urine volume. If the output was at or above the augmentation limit then the next specimen receiving the full effects of the infusion would be well above the augmentation limit. With this knowledge the time of the first catheterization following the infusion would be very accurately taken and a blood specimen obtained before the next catheterization. The dogs were catheterized every fifteen minutes because with a marked diuresis, as that produced in this experiment, the urine volume changes very rapidly, increasing at first and decreasing later. If the urine was collected over a long period of time, it would be difficult to be sure that the urine volume during the entire time was above the augmentation limit. In other words, if the urine was collected over a period of an hour, as advocated by Moller et al.,[1] the originators of the urea clearance test, the excretion rate might be only 1 c.c. a minute for the first and last portions of the hour, but 3 to 4 c.c. a minute during the middle of the hour. The total volume of urine excreted for the hour would average more than 2 c.c. a minute, but since the urine volume fell below the augmentation limit for part of this time there would not have been the optimum kidney function and the maximum amount of urea would not have been excreted. The variation in the urine volume during a fifteen minute period would not be sufficiently great to allow this error to be made.

Collection of the specimen of urine every quarter of an hour further simplifies the experiment by permitting catheterization before the bladder becomes full. When larger quantities of urine were allowed to accumulate in the dogs' bladder, it was almost impossible to prevent the dogs from voiding before the specimen could be collected. The urine volume was measured in tenths of a cubic centimeter, and time on a ten second scale. When the specimens were taken in this manner the variation in the maximum urea clearance values during several successive fifteen minute periods was found to be within the limit of error for the procedure, i.e. 3 per cent.

The urea determinations were done by the urease aeration method. All of the specimens were collected in the late morning and the determinations made during the afternoon of the same day. The dogs were fed only in the afternoon and always following the collection of the specimen. The animals received from 1 to 2 pints of Miller's A-1 ration a day, which food contains about 12 per cent protein. Every Saturday the dogs were given meat. They were kept in individual cages, where it was possible to take but very little exercise. It has been shown that the urea clearance, both maximum and standard, is markedly decreased by exercise.

Six dogs were used and the observations made over a period of from three to four weeks. A total of fifty determinations were made. On each dog one or more normal readings were made and the dogs submitted to nephrectomy. The right kidney was always removed through a midline incision. Urea clearance determinations were made on about the second or third day following operation and every two or three days after this for a period of several weeks. The kidney removed at operation and that obtained at autopsy after sacrificing the animal were weighed, their dimensions taken and their volume measured. These measurements were made as checks in order to prevent any gross error in recording these values.

The following is a representative protocol:

Dog H5. Black and tan cur. Weight 6.9 kg.

August 4, 1931.

9:25 A.M. 500 c.c. water given by stomach tube.

10:10. Catheterized. Specimen not saved.

10:04. Infusion 150 c.c. normal saline.

10:25. Catheterized. Specimen not saved.

10:40. Catheterized; 60 c.c. urine saved. Labeled Specimen A.

10:43. 10 c.c. blood taken. Labeled Specimen B.

10:55. Catheterized. 55 c.c. urine saved. Labeled Specimen C.

11:05. 10 c.c. blood taken. Labeled Specimen D.

11:10. Catheterized. 54 c.c. urine saved. Labeled Specimen E.

Specimen A contained 71.4 mg. of urea per 100 c.c.

Urine volume 4 c.c. per minute.

Specimen B contained 11.04 mg. of urea per 100 c.c.

Urea clearance for this determination 25.8 c.c. blood.

Specimen C contained 81.0 mg. of urea per 100 c.c.

Urine volume 3.66 c.c. per minute.

Specimen D contained 12.24 mg. of urea per 100 c.c.

Urea clearance for this determination 23.4 c.c. blood.

Specimen E contained 82.2 mg. of urea per 100 c.c.

Urine volume 3.6 c.c. per minute.

Urea clearance using specimen D, 24.1 c.c. blood.

Average urea clearance 24.4 c.c. blood.

Average blood urea concentration 11.64 mg. per 100 c.c.

August 6, 1931. The animal was operated upon under ether anesthesia, and the right kidney removed through a midline incision. The kidney was found to weigh 26 gm. Its measurements were 5.3 by 3.4 by 2.5 cm. Its volume by water displacement was 25 c.c.

August 8, 1931. The dog seems to be in excellent condition, apparently fully recovered from the immediate effects of the operation.

Urea concentration of urine 129 mg. per 100 c.c.

Urine volume 2 c.c. per minute.

Blood urea concentration 11.66 mg. per 100 c.c.
Maximum urea clearance for this determination 22.2 c.c. blood.
Urea concentration of urine 103.5 mg. per 100 c.c.
Urine volume 2.4 c.c. per minute.
Blood urea concentration 12.39 mg. per 100 c.c.
Urea clearance for this determination 20.0 c.c. blood.
Average urea clearance 21.1 c.c. blood.
Average blood urea concentration 12.02 mg. per 100 c.c.

August 10, 1931.
Urea concentration of urine 156.9 mg. per 100 c.c.
Urine volume 2.4 c.c. per minute.
Blood urea concentration 23.28 mg. per 100 c.c.
Urea clearance 16.1 c.c. blood.

August 12, 1931.
Urea concentration of urine 64.5 mg. per 100 c.c.
Urine volume 2.8 c.c. per minute.
Blood urea concentration 12.96 mg. per 100 c.c.
Urea clearance 13.9 c.c. blood.

August 14, 1931.
Urea concentration of urine 85.5 mg. per 100 c.c.
Urine volume 2.87 c.c. per minute.
Blood urea concentration 14.4 mg. per 100 c.c.
Urea clearance 17.0 c.c. blood.

August 17, 1931.
Urea concentration of urine 153 mg. per 100 c.c.
Urine volume 2.49 c.c. per minute.
Blood urea concentration 25.2 mg. per 100 c.c.
Urea clearance 15.1 c.c. blood.

August 20, 1931.
Urea concentration of urine 73.5 mg. per 100 c.c.
Urine volume 2.62 c.c. per minute.
Blood urea concentration 16.1 mg. per 100 c.c.
Urea clearance 11.9 c.c. blood.

August 24, 1931.
Urea concentration of urine 330.0 mg. per 100 c.c.
Urine volume 3.26 c.c. per minute.
Blood urea concentration 63.0 mg. per 100 c.c.
Urea clearance 17.0 c.c. blood.

August 27, 1931.
Urea concentration of urine 93.0 mg. per 100 c.c.
Urine volume 2.84 c.c. per minute.
Blood urea concentration 16.8 mg. per 100 c.c.
Urea clearance 15.7 c.c. blood.

August 31, 1931.
Animal sacrificed. Body weight 6.6 kg. Body volume 3.6 liters. The left kidney was removed and found to be macroscopically normal. Kidney weight 21 gm. Volume (by water displacement) 21 c.c. The kidney measured 4.9 by 3.4 by 2.4 cm.

BODY WEIGHT AT BEGINNING AND END OF EXPERIMENT

The dogs were weighed at the beginning of the experiment and again at the end immediately after they were sacrificed. They all lost weight with the exception of one animal whose weight remained the same. The maximum loss was 2.4 kg. The average loss was 0.8 kg., or 9 per cent of the average original weights. (Table 1.)

TABLE 1
BODY WEIGHT AT THE BEGINNING AND END OF EXPERIMENT

Dog	Beginning of Experiment (Kg.)	End of Experiment (Kg.)	Difference (Kg.)
H5	6.9	6.6	0.3
H6	6.2	6.0	0.2
H7	6.7	5.3	1.4
H8	12.5	10.1	2.4
H9	10.8	10.1	0.7
H10	6.4	6.4	0.0
Average	8.2	7.4	0.8

RELATION OF BODY WEIGHT TO RENAL WEIGHT

The weight of the dogs varied from 6.2 to 12.5 kg. The average was 8.2 kg. The renal weight varied from 37 to 65 gm., with an average of 48.5 gm. Compensatory hypertrophy was not taken into consideration. The amount of renal tissue per kilogram body weight was fairly constant, varying from 4.7 to 6.8 gm. with an average

of 6.0 gm., and a variability of 13 per cent. (Table II.)

TABLE II

RELATION OF BODY WEIGHT TO RENAL WEIGHT

Dog	Body Weight (Kg.)	Renal Weight (Gm.)	Renal Weight per Body Weight (Gm./kg.)	Renal Weight per Body Weight; Variability from Average (Per Cent)
H5	6.9	47	6.8	12
H6	6.2	38	6.1	2
H7	6.7	45	6.7	12
H8	12.5	59	4.7	13
H9	10.8	65	6.0	0
H10	6.4	37	5.7	11
Average	8.2	48.5	6.0	

WEIGHT OF RENAL TISSUE AT BEGINNING AND END OF EXPERIMENT

During this experiment both the renal weights and volumes were taken, but since the figures were so nearly the same and since the determinations reported in the literature have used the weights as a standard, this custom was adhered to in this paper.

Addis et al[7] found that in 22 rabbits the weight of both kidneys was approximately the same, there being an average difference of 2.1 per cent.

Using the data of Hinman[8] on compensatory hypertrophy in the rat, Addis et al.[7] calculated an 8 per cent increase in the renal weight between fifteen and thirty-three days after nephrectomy, estimating the renal weight as 58 per cent of the original value at this time. At the end of their experiment, 106–126 days following nephrectomy, the weight of renal tissue per kilogram body weight was found to be 66 per cent of the prenephrectomy value. This observation was made on 6 rabbits. Hinman[8] obtained an average of 20 per cent total increase in the renal weight after a unilateral ureteral ligation. He says that in no case was the size of the kidney doubled nor did the increase ever even approach this degree.

In normal kidneys the weight can be taken as a fairly accurate index of the amount of functioning renal tissue, but this relation is disturbed after compensa-

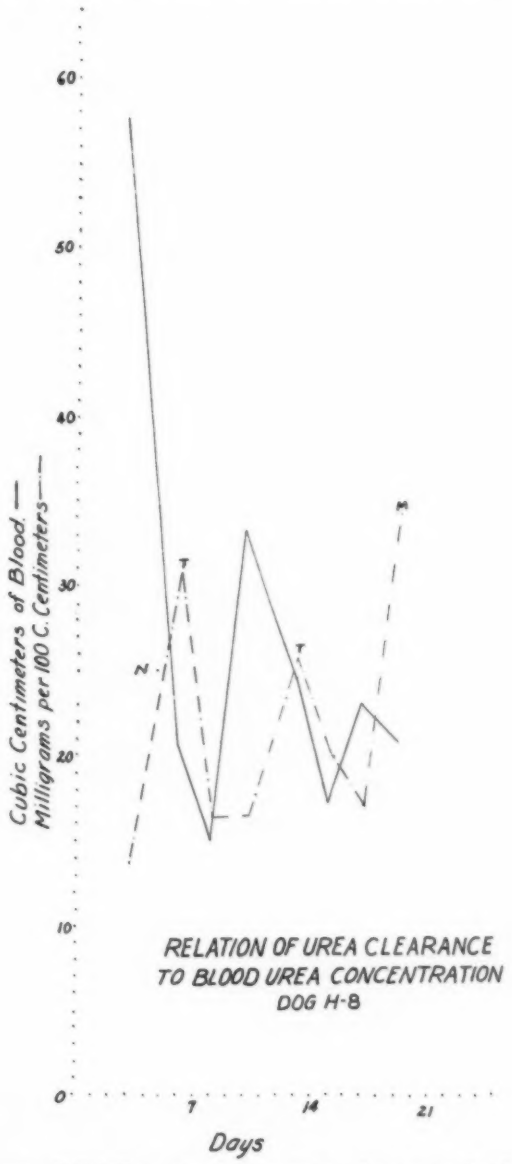

FIG. 4. Solid line, maximum urea clearance; broken line, blood urea concentration; N, nephrectomy; T, Tuesday; M, Monday.

In this animal the renal weight was reduced to 55 per cent and the renal function to 40 per cent of the prenephrectomy values. The greatest reduction in renal function, as measured by the urea clearance, was recorded in this animal. The subsequent variation in this factor is seen to be much more marked than in the other animals. This is the only animal in which the variation in the urea clearance exceeded the variation in the blood urea concentration.

tory hypertrophy as has been shown by Oliver,[9] because of a difference in the degree of hypertrophy of the several elements of the kidney. This explains the discrepancy obtained when the hypertrophied renal weight is compared to the renal function after nephrectomy.

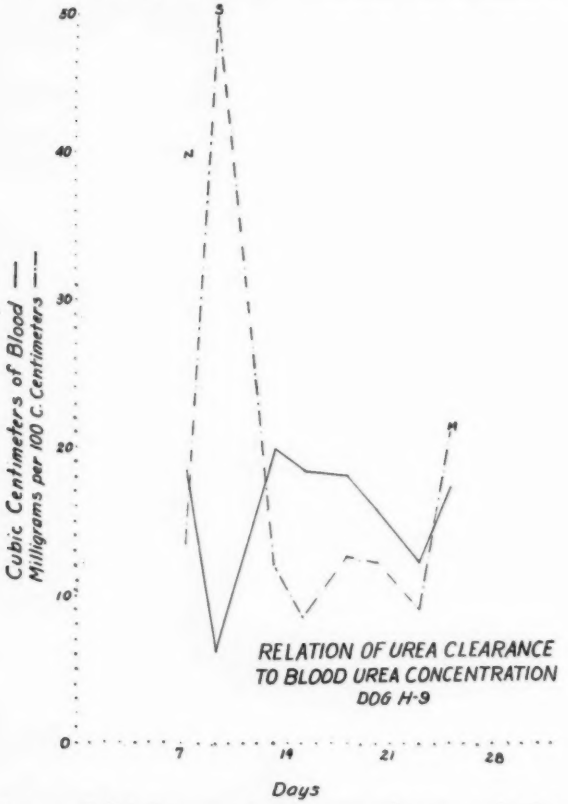

FIG. 5. Solid line, maximum urea clearance; broken line, blood urea concentration; N, nephrectomy; S, Saturday; M, Monday.

The results in this animal are unusual because of the rapid return of the clearance values to normal after nephrectomy. It would seem that the remaining kidney had little difficulty in assuming most of the function of the lost kidney in this animal. The average renal function, as measured by the maximum clearance, for the first week after nephrectomy amounts to 79.5 per cent of the prenephrectomy value. The renal weight was reduced to 56 per cent. The large increase in the blood urea concentration on Saturday might be attributed to the nephrectomy which was done the day before. There is no rise in the blood urea concentration on Monday in this animal, with the exception of a slight increase at the last observation. This is in keeping with the good renal function as indicated by the urea clearance.

In the experimental work reported by the author the average weight of the right kidney was 22 gm. and the average weight of the left kidney obtained two weeks later at autopsy was 26.5 gm. There was an average difference of 4.5 gm. This difference cannot be entirely attributed to compensatory hypertrophy because in dog H5 the right kidney weighed 5 gm. more than the left in which the compensatory hypertrophy was supposed to have occurred. This demonstrates the fallacy in the assumption that both kidneys weighed the same before nephrectomy. In calculating the total renal weight and the reduction by nephrectomy the actual weights of the two kidneys were used and no compensatory hypertrophy taken into consideration. This would seem justifiable in view of the discrepancy between the weights of the right and left kidneys. While some hypertrophy probably occurred it is impossible to say how much. The reduction in renal weight for the entire series is shown in Table III.

TABLE III

RENAL WEIGHTS BEFORE AND AFTER NEPHRECTOMY

Dog	Total Kidney Weight before Nephrectomy (Gm.)	Renal Weight after Nephrectomy (Weight of Left Kidney) (Gm.)	Reduction of Renal Weight (Per Cent)	Weight of Right Kidney (Gm.)
H5	47	21	44	26
H6	38	23	60	15
H7	45	24	53	21
H8	59	33	55	26
H9	65	37	56	28
H10	37	21	56	16
Average	48.5	26.5	54.6	22

RELATION OF RENAL WEIGHT, BODY WEIGHT AND BODY VOLUME TO UREA CLEARANCE BEFORE NEPHRECTOMY

It would seem that if there is a definite relation between the amount of functioning renal tissue and the urea clearance values there should be a direct relation between the renal weight, the body weight or body volume and the urea clearance in a series of dogs. These comparisons were made in six dogs and the results are shown in Tables

IV and V.

The variation of 72 per cent in the clearance per gram of renal tissue would seem to indicate either that there are marked differences in the functioning ability of 1 gm. of renal substance or that there are certain unknown factors which markedly affect the action of this gram of

TABLE IV

RELATION OF UREA CLEARANCE TO RENAL AND BODY WEIGHTS AND BODY VOLUME

Dog	Body Weight (Kg.)	Body Volume (Liters)	Total Kidney Weight (Gm.)	Prenephrectomy Urea Clearance (C.c.)
H5	6.9	3.6	47	24.4
H6	6.2	4.3	38	21.9
H7	6.7	5.0	45	27.1
H8	12.5	9.5	59	57.4
H9	10.8	9.7	65	18.6
H10	6.4	3.9	37	16.1
Average	8.2	6.0	48.5	27.6

renal tissue. While it is possibly a coincidence it is still interesting to note that the body weight and volume more nearly parallel the urea clearance than does the kidney weight. In the final analysis this series is small and it cannot be said that

TABLE V

RELATION OF UREA CLEARANCE TO UNITS OF RENAL AND BODY WEIGHTS AND BODY VOLUME

Dog	Urea Clearance per Kilogram Body Weight (C.c.)	Urea Clearance per Liter Body Volume (C.c.)	Urea Clearance per Gram Kidney Weight (C.c.)
H5	3.53	6.77	.519
H6	3.53	5.09	.576
H7	4.04	5.42	.602
H8	4.58	6.04	.972
H9	1.72	1.91	.286
H10	2.51	4.12	.436
Average	3.31	4.89	.565
Deviation −	1.59	2.98	.279
Deviation +	1.27	1.88	.407
Variability	48 per cent	60.9 per cent	72 per cent

these figures are conclusive but they might be considered significant. In the work of Addis et al[7] demonstrating the relation of the dimunition in the value of the ratio

$$\frac{\text{Urea in one hour's urine}}{\text{Urea in 100 c.c. blood}}$$

and the diminution in the amount of renal tissue following nephrectomy, the ratio was calculated in terms of kilogram body weight. The determinations were made on 6 rabbits. The urea excretion ratio or maximum clearance was found by Taylor, Drury and Addis[2] to more nearly parallel the body surface area than the body weight.

The greatest deviation from the average in the urea clearance per gram renal weight was in dog H8 which incidentally had the highest clearance values and the smallest amount of renal tissue per kilogram body weight. It would seem that the kidneys in this dog were far more efficient than the average because the clearance value was .972 c.c. of blood per gram renal tissue while the average clearance was only .565 c.c. of blood per gram renal tissue. This dog also had the most efficient kidneys when the clearance was compared to the total body weight, i.e. there were 4.58 c.c. of blood cleared per kilogram body weight when compared with the total average of 3.31 c.c. of blood per kilogram body weight. The urea clearance values for this dog far exceeded those for any other dog. The urea clearance values for H8 before nephrectomy were checked on two successive days because they were so much higher than the values from the other dogs. Determinations made from specimen drawn at fifteen minute intervals always checked within 3 per cent, therefore only one determination for the values before nephrectomy is listed.

RATE OF UREA EXCRETION

In this experiment there was a decided fall in the maximum urea clearance values following nephrectomy. There was no constant rise in the rate towards the end of the two weeks period of observation. In four of the dogs the average value

seemed to remain about the same after the initial fall. In dog H9 the urea clearance had arisen to above normal at the second

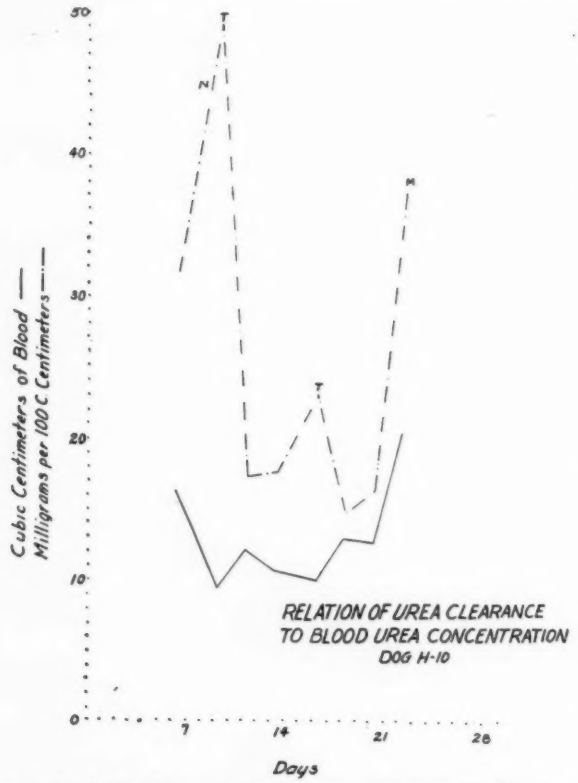

Fig. 6. Solid line, maximum urea clearance; broken line, blood urea concentration; N, nephrectomy; T, Tuesday; M, Monday.

The blood urea concentration in this animal undergoes a marked decrease at the second observation following nephrectomy and remains below the prenephrectomy value until the final observation. The maximum clearance before nephrectomy was lowest in the series and the variation after nephrectomy was also less than in any other animal. The maximum clearance value following nephrectomy exceeded the prenephrectomy reading in 2 of 50 observations. The last determination for this animal was one of these two instances. Nephrectomy reduced the maximum clearance to 65.8 per cent and the renal weight to 56 per cent of the prenephrectomy values in this animal. The increase in the blood urea concentration is seen to occur during the first of the week in this animal.

observation after nephrectomy and then proceeded to decline gradually. In dog H10 there was a sudden marked rise above the prenephrectomy value on the last day of observation. In some cases the postnephrectomy fall in the clearance values was greater than in others, it was always smaller in cases that had low clearance values before nephrectomy. The variation after nephrectomy was also always less when the original urea clearance was small. As has been said before, the ratio of Addis, when the standard conditions set forth for determining it are used, closely resembles the maximum clearance. Addis et al.[7] found a reduction in the rate of urea excretion from 100 to 68.6 per cent in from fifteen to thirty-three days after nephrectomy, while the estimated amount of renal tissue for this time was 58 per cent. This difference of 10 per cent was very much smaller than when any other functional method was used. An attempt to parallel the amount of functioning kidney tissue and the urea clearance values in this experiment met with fair success. To simplify matters in this investigation the urea clearance values before and after nephrectomy were compared to the weights of the two kidneys or the weight of the renal tissue before and after nephrectomy. No compensatory hypertrophy was taken into consideration, because the weights of the kidneys varied to such an extent that an estimation would be very difficult and rather uncertain. If an allowance had been made for hypertrophy in the remaining kidney, the percentage of reduction of the function would more nearly approach the percentage of reduction of the weights. Using the weight of both kidneys as the total renal weight or 100 per cent, after nephrectomy the average renal weight was 54.6 per cent. The average urea clearance value for the first three determinations after nephrectomy, covering a period of about one week, was 56.2 per cent of the original value. This is a very close approximation of the two values, but if each dog is considered the results appear quite different. In only two out of the six cases did the reductions correspond within 3 per cent. In the other four instances there was a difference in from 9.2 to 26 per cent. The reduction in the renal weight of dog H8 was 15 per cent greater than the reduction in the renal function, whereas in four other dogs the greatest reduction was in the

opposite direction, the reduction in renal function being from 3 to 26 per cent greater than the reduction in the renal weight. (See Tables VI and VII.) The averages 56.2

Table VI

MAXIMUM UREA CLEARANCE BEFORE AND AFTER NEPHRECTOMY

Dog	Maximum Urea Clearance before Nephrectomy (C.c.)	Maximum Urea Clearance after Nephrectomy (Average of First Three Observations) (C.c.)	Reduction (Per Cent)
H5	24.4	17.1	70.0
H6	21.9	13.8	63.0
H7	27.1	13.8	50.9
H8	57.4	23.0	40.0
H9	18.6	14.8	79.5
H10	16.1	10.6	65.8
Average	27.6	15.51	56.2

and 54.6 were calculated as percentages from the average values of the renal weight and function before and after nephrectomy.

Table VII

COMPARISON IN REDUCTION OF RENAL WEIGHT AND FUNCTION

Dog	Reduction in Renal Function (Per Cent)	Reduction in Renal Weight (Per Cent)	Difference (Per Cent)
H5	70.0	44	26.0
H6	63.0	60	3.0
H7	50.9	53	2.1
H8	40.0	55	15.0
H9	79.5	56	23.5
H10	65.8	56	9.8
Average	56.2	54.6	1.6

It would seem then that the occurrence of a difference of only 1.6 per cent between the average reduction of renal function and weight is more of a coincidence than proof of the value of this functional test for determining the amount of functioning renal tissue.

There was no constant day-to-day variation in the urea excretion. No definite upward trend could be noticed during the two or three week period of observation following nephrectomy. The urea clearance value reached a point higher than the prenephrectomy reading in 2 cases. The average clearance value for the entire period of observation following nephrectomy was only 0.4 c.c. greater than the average value for the first week after nephrectomy. The variation in the clearance value following nephrectomy was rather marked and this could be neither understood nor controlled.

BLOOD UREA CONCENTRATION

It is the general consensus of opinion that a reduction of 50 per cent in the renal weight causes no definite increase in the blood urea concentration under normal conditions. Addis et al.[7] believe the increase in the blood urea concentration observed by Holweg after unilateral nephrectomy and by Addis and Watanabe after ligation of one ureter were due to the increased protein catabolism during the first few days following such operations. Addis et al.[7] believe that under conditions of strain the decrease in the renal tissue from unilateral nephrectomy may lead to a slight increase in the blood urea concentration. In the experiment reported here an interesting observation was unwittingly made, and while the procedure was not sufficiently controlled to warrant definite conclusions, the results are suggestive. As can be readily seen from the graphs there was no constant persistent postoperative rise in the blood urea concentration. While the urea clearance observations are uniformly less several days after operation, the blood urea concentrations either have the same value or are above or below the prenephrectomy readings. It is interesting to note the marked rise in the blood urea concentration during the first part of the week. The dogs were fed meat every Saturday afternoon and always after the observations for that day were made. While no prenephrectomy determinations were made during the first part of the week, such a rise could hardly be expected to be called normal. It is also interesting

to note that under this strain, while the blood urea concentration is markedly increased the urea clearance is not uniformly affected if at all. The results are only suggestive, as has been said, that an increase in the blood urea concentration occurs following nephrectomy when a strain is put on the kidneys.

The estimates of the urea clearance and blood urea concentration were observed over a period of three weeks, but no definite variation from the immediate postnephrectomy values could be made out. The average value for all of the postnephrectomy urea clearance observations was 15.9 c.c. This covered a period of about three weeks or over. The average value for the first week after nephrectomy was 15.5 c.c. This difference of 0.4 c.c. can not be considered significant.

DISCUSSION

It must be admitted at the outset that the series of animals used in this experiment is too small for positive conclusions to be drawn. The results, however, are sufficiently positive to be considered significant and should serve to further strengthen the confidence in the maximum urea clearance test as an accurate determination of kidney function.

The variation in the relation of the renal weight to the unit of body weight demonstrates the folly of attempting to correlate the two. There was a variation of as much as 13 per cent from the average and in only two instances was the variability under 2 per cent.

Compensatory hypertrophy was not considered in this experiment. If the figures of Hinman[8] on hypertrophy in the rat are accepted, the compensatory renal enlargement during this two week postnephrectomy period would be small, at least under 8 per cent. There is no accurate means of determining the amount of hypertrophy and an uncertain calculation would greatly complicate the results.

A considerable variation in the urea clearance per unit of body weight, body volume and renal weight was found. This can be understood, however, when the variation in the relative proportions of body weight, body volume and renal weight in one animal is considered. If a unit of renal tissue is capable of a definite amount of work, there should be a close similarity in the function per gram of renal tissue in the several dogs. In this experiment the variability in this factor amounted to 72 per cent. It is a well-established fact that nephritis is very common in dogs. It is possible that some of the ainmals used in this experiment had had a nephritis and the renal weight was diminished below the normal. The renal function would not necessarily be disturbed because it might have returned to normal with the unequal hypertrophy of renal tissue as was shown to occur by Oliver.[9] It is possible that the renal function was not normal in several of the dogs and this would help account for the rather marked variation in the clearance per unit of renal weight.

A maximum variation of 26 per cent and an average of 1.6 per cent between the reduction in renal weight and function after nephrectomy would seem to indicate from these studies that a direct relation between the kidney weight and function cannot be made in the dog. The relation is sufficiently close, however, to obtain a general idea of the functioning renal tissue from a maximum urea clearance determination. These results would support other work on the urea clearance in establishing its worth as a test of kidney function. These results would seem to contradict the statement commonly made that unilateral nephrectomy has no appreciable effect on the various kidney function tests employed in estimating the renal functional capacity.

In this experiment the blood urea concentration after nephrectomy remained around normal during the latter part of the week, but became elevated during the first portion of almost every week. This variation was attributed to a high protein intake Saturday afternoon. On this day,

after the observations were made, the animals were fed meat in addition to their regular diet of Miller's A-1 ration. This factor was not sufficiently controlled to warrant definite conclusions, but was interpreted as meaning that a definite rise in the blood urea concentration occurred after unilateral nephrectomy only when a strain was put on the kidneys.

CONCLUSIONS

The maximum urea clearance, which is a ratio of urea excretion, is an accurate means of measuring kidney function. If we accept the consensus of opinion among medical authors of the value of various other renal function tests, it would seem that the maximum urea clearance is far superior to any other known method of measuring renal function. Exception to this is made for the standard urea clearance, which is equivalent to the maximum clearance for smaller volumes of urine, and the ratio of Addis,

$$\frac{\text{Urea in one hour's urine}}{\text{Urea in 100 c.c. blood}},$$

when the standard conditions for determining it are used. This ratio has been shown to be very similar to the maximum urea clearance.

The series of animals used in these observations is too small for positive conclusions to be drawn but the results check themselves sufficiently to be considered significant.

The maximum urea clearance was compared to the units of body weight, body volume and kidney weight. Average values were calculated. The average clearance per kilogram body weight was 3.31 c.c. of blood and the variability of this figure 48 per cent. The average clearance per liter body volume was found to be 4.89 c.c. of blood and the variability 60.9 per cent. The average clearance per gram of renal tissue was 0.565 c.c. of blood and the variability 72 per cent. The possibility of an old nephritis with an uneven compensatory hypertrophy was offered as an explanation for the marked variation in the relation of the urea clearance to the renal weight.

In the dog a definite relation between the reduction in the maximum clearance and the reduction in the renal weight following nephrectomy could not be established.

There was no appreciable difference between the immediate postnephrectomy maximum urea clearance or blood urea concentration and the observations made two weeks after nephrectomy.

There was no constant persistent postoperative rise in the blood urea concentration. This value was much greater than normal in practically every observation made on Monday and frequently in those made on Tuesday. This was attributed to a high protein diet Saturday afternoon following the observations for that day. This discovery was believed to indicate a diminution in the ability of the kidney to handle urea in large quantities.

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NEWER CONCEPTS ON THE PHYSIOPATHOLOGY AND TREATMENT OF THROMBOANGIITIS OBLITERANS

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WHILE the outstanding lesion in thromboangiitis obliterans is a thrombotic closure of both large and small vessels of the extremities, especially the lower, many of the clinical features of the disease cannot be attributed solely to this lesion. Much more intricate and complex changes in the body organism seem to underlie the life cycle of the tissues affected.

The early disturbances in gas exchange between capillaries and surrounding tissues as evidenced by rubor, the not uncommon vasomotor irritability simulating Raynaud's disease, the capillary bed paralysis with resulting stasis and thrombosis and not infrequently capillary endovasculitis, are phenomena quite independent of the classical pathognomonic architectural changes seen in the large and small vessels of the affected part. There seems to be a preponderance of evidence that the factor or factors to be reckoned with in the production of thrombosis of the large and small vessels of the extremities are also directly responsible for the production of the aforementioned observations. Since the thrombotic tendency of this baffling disease figures preeminently as the most striking feature, it is plainly evident that changes in the blood chemistry affecting the coagulability of blood may explain the physiopathology of the disease. In addition to the calcium content, the phospholipins, especially the lecithins, occupy an important place in the processes affecting the coagulability of blood. Woolridge (1893) showed that the alcohol ether extract of red blood corpuscles had a marked effect in inducing coagulation. Serum deprived of its lecithin would only coagulate after the latter had been again added to it. Matthews follows Woolridge and considers the clotting of the blood as essentially the crystallization of the phospholipin protein compound, i.e., blood plasma, the stability of which is easily upset in many ways. Howell, in 1912, went a step further and showed that kephalin was the active thromboplastic substance. From the aforesaid evidence, however, there is a general agreement among investigators, that phospholipins (lecithin) are concerned in producing a thromboplastic effect, the withdrawal of which decreases and the addition increases coagulability. Were the phospholipins not essential in the metabolism of life, it would be almost impossible to understand their invariable presence whenever the phenomena of life are present. That they are indispensable is obvious, but the particular part they play in the pathological entity known as thromboangiitis obliterans has opened up a new field of investigation.

Upon this basis, a study of the lecithin content of the whole blood and plasma was undertaken on a series of 50 cases of thromboangiitis obliterans. In the same series and under similar conditions, morning urines were examined for choline, a split product of lecithin metabolism (Table I). In order to eliminate a possible source of contention as to normal values obtained by various investigators, we have undertaken to assess a standard lecithin value for whole blood and plasma by the method of Bloor, Benedict and Theis on 25 males free from any evidence of thromboangiitis obliterans (Table II). Under similar conditions morning urines were examined for choline. From the figures, (Table I) it is very apparent that there is a marked increase in the lecithin content of the whole

TABLE 1

LECITHIN CONTENT OF THE BLOOD IN TERMS OF MILLIGRAMS OF ORGANIC PHOSPHORUS PER 100 C.C. SERIES OF 50 CASES OF THROMBOANGIITIS OBLITERANS

Patient	Date	Whl. Bl.	Plasma	Urine Choline	Comments	
					Condition	Result
M. S.....	12/ 3/29	11.0	....	pos. ++++	Ulcer left ring finger, gangrene 4th toe (right)	Healed
	12/28/29	10.0	....	pos. ++++		
	11/15/30	15.0	....	negative		
	4/ 7/31	13.7	....	negative		
M. W.....	12/ 5/29	9.5	....	pos. ++++	Ulcer, 5th toe (left)	Healed
	11/22/30	11.0	7.9	pos. ++++		
	3/31/31	15.0	11.9	negative		
S. S.....	12/ 6/29	10.0	....	pos. ++++	Ulcers, all toes (right)	Healed
	11/17/30	11.0	7.6	pos. ++++		
	11/14/31	11.0	8.4	pos. ++++		
J. S.....	1/ 8/29	9.0	....	pos. ++++	High amputation (left) Gangrene of foot (right)	Healed met. ph. stump
	4/ 8/31	13.3	11.1	negative		
A. R.....	1/28/29	12.0	....	pos. ++++	Gangrene of foot up to ankle (left)	Healed met. ph. stump
	2/19/29	11.0	....	pos. ++++		
	3/11/29	11.0	....	pos. ++++		
	4/28/29	10.0	....	pos. ++++		
	11/22/30	12.0	7.0	negative		
	4/11/31	10.5	8.0	negative		
B. S.....	2/ 3/30	12.5	....	pos. ++++	Ulcers, 1st & 2nd toes (left)	Healed
	2/19/30	10.0	....	pos. ++++		
	3/26/30	7.0	....	negative		
B. P.....	2/ 5/30	12.5	....	negative	Ulcer, 3rd toe (right)	Healed
	3/30/30	10.0	....	pos. ++++		
	11/17/30	10.0	....	pos. ++++		
	4/ 4/31	12.0	7.7	negative		
I. W.....	4/11/30	9.0	....	pos. ++++	Ulcer, 1st toe (right)	Improved
	11/20/30	11.1	8.0	negative		
S. W.....	3/17/30	13.5	....	negative	Claudication and rubor (both feet)	Improved markedly
	11/14/30	10.0	....	pos. ++++		
	4/ 8/31	14.0	10.0	negative		
B. G.....	5/ 7/30	9.0	....	pos. ++++	Gangrene 1st & 2nd toes (right)	Healed
	11/14/30	13.6	....	negative		
M. E.....	5/24/30	8.5	....	pos. ++++	Claudication and rubor	Improved markedly
	11/14/30	9.10	....	negative		
L. F.....	5/21/30	9.2	....	pos. ++++	Gangrene of foot to ankle (right)	Healed met. ph. stump
	1/ 9/31	12.8	8.0	negative		
	4/ 9/31	10.7	9.0	negative		
H. N.....	11/13/30	11.3	....	pos. ++++	Claudication and rubor (both feet)	Improved markedly
	4/14/31	12.0	10.0	negative		
N. B.....	6/27/30	....	....	pos. ++++	Claudication and gangrene 5th toe (right)	Improved markedly
	11/20/30	13.0	12.5	negative		
M. S.....	11/15/30	14.5	....	pos. ++++	Claudication and rubor (both feet)	Improved markedly
	4/ 4/31	14.7	6.3	negative		

TABLE 1 (*Continued*)

Patient	Date	Whl. Bl.	Plasma	Urine Choline	Comments	
					Condition	Result
D. F	1/ 3/31 4/ 4/31	12.0 11.0	9.0 10.0	pos. ++++ negative	Claudication and rubor (left)	Improved markedly
J. G	1/20/31	12.4	11.0	negative	Claudication and rubor (both feet)	Improved markedly
J. B	2/25/31	13.0	11.5	negative	Claudication and rubor (left)	Improved markedly
L. L	10/14/31	12.2	9.8	pos. ++++	High amputation (right)	Untreated
H. W	7/ 2/30 4/ 5/31	11.0 12.1	9.1 8.3	pos. ++++ negative	Claudication and rubor (both feet)	Improved markedly
B. O. (F)	3/23/31	12.6	8.0	negative	Claudication and rubor (left)	Untreated
D. S	4/11/31	10.6	8.1	pos. ++++	Healed ulcers 1st & 2nd toes (right)	Untreated
S. S	1/ 6/31	13.0	11.0	negative	Amputation (both thighs)	Untreated
M. S	1/ 6/31	14.0	9.8	negative	Amputation (both thighs)	Untreated
H. W	11/29/30	12.0	9.0	pos. ++++	Ulcers, 2nd and 3rd toes (right)	Untreated
F. C	4/14/31	13.0	7.5	negative	Gangrene 1st & 3rd toes (right)	Healed
H. P	4/21/31	12.8	9.0	negative	Gangrene 1st toe (left)	Healed
B. L	5/21/31 7/21/31	13.2 12.4	9.6 9.0	pos. ++++ negative	Gangrene 1st & 2nd toes (left)	Healed
D. M	5/16/31 6/ 3/31	11.6 13.0	8.3 10.1	pos. ++++ negative	Gangrene 1st toe (left)	Untreated
E. S	6/ 2/31	11.1	9.0	negative	Healed ulcer 1st toe (right) rubor	Improved markedly
S. F	7/ 8/31 11/16/31	12.8 13.8	10.0 10.9	negative negative	Claudication and rubor (left)	Improved markedly
L. W	3/ 6/30 11/17/30 4/ 9/31	10.5 13.8 11.2	.... 9.3	pos. ++++ negative negative	Ulcers 1st, 2nd & 3rd toes (right) 1st toe (right)	Healed
H. L	5/12/30 4/ 5/31	14.8 12.0	.... 10.1	pos. ++++ pos. ++++	Gangrene foot (right) Arteriosclerotic changes	Amputation
Z. K	11/24/30	12.5	11.3	negative	Claudication and rubor (both feet)	Improved
P. T	2/ 5/31	14.5	10.0	negative	Claudication and rubor (both feet)	Improved
L. B	5/ 1/31	13.2	9.0	negative	Gangrene 4th toe (left) rubor	Healed
M. G	7/23/31	12.5	9.3	negative	Gangrene foot (left) Arteriosclerotic changes	Amputation

Table 1 (*Continued*)

Patient	Date	Whl. Bl.	Plasma	Urine Choline	Comments	
					Condition	Result
J W.....	11/ 9/31	12.5	10.4	pos. ++++	Ulcer, 4th toe (right)	Healed
I. H.....	7/14/31	14.5	9.5	negative	Amputation (both thighs)	Untreated
J. F.....	8/19/31	12.5	10.2	negative	Gangrene of foot to ankle (right)	Demarcating met. ph. joint
I. T.....	6/13/31 8/19/31	11.3 12.0	10.0 10.0	pos. ++++ negative	Claudication and rubor (both feet)	Improved
H. P.....	10/20/31 11/19/31	13.4 13.5	10.2 10.1	negative negative	Gangrene of foot to ankle (left)	Demarcating met. ph. joint
D. S.....	11/ 6/31	14.5	11.2	negative	Amputation thigh (right)	Untreated
M. W.....	11/ 9/31	11.0	9.0	pos. ++++	Pallor (both feet) absent "dorsalis pedis"	Improved
S. B.....	11/16/31	13.2	10.0	pos. ++++	Claudication, rubor phlebitis (right)	Improved
D. F.....	11/19/31	11.8	8.2	pos. ++++	Amp. thigh (left) Ulcers, toes (right)	Improved
M. U.....	11/19/31	12.5	8.7	negative	Gangrene foot (left) Arteriosclerotic changes	Amputation
J. G.....	12/ 1/31	14.0	10.9	negative	Amputation thigh (right) rubor & pain (left)	Improved
M. B.....	12/16/31	13.6	10.5	pos. ++++	Claudication and rubor	Improved
D. L.....	5/27/31	13.9	10.8	negative	Gangrene foot (right) Arteriosclerotic changes	Amputation

The normal average lecithin values for whole blood evaluated was 11.0 and for plasma 8.2. In the series of 50 cases of thromboangiitis obliterans, 44 or 88 per cent showed a whole blood lecithin value of 12.0–14.7; 43 or 80 per cent of 54 whole blood lecithin values above 12.0 gave a negative reaction to choline in the urine. Thirty-two or 86 per cent of 37 whole blood lecithin values below 12.0 showed a very marked choline reaction in the urine. All cases showed a choline reaction in the urine at some time while under observation.

blood in thrombic angiitis obliterans. The plasma with but few exceptions shows a proportionate increase. At this point it is interesting to note that 3 cases of thrombocytopenic purpura in which a thrombotic tendency would have been welcome, showed a very low blood lecithin value.

The viscosity or internal friction of the blood plays an important role as a contributory factor in the production of thrombosis. Decreasing it makes the blood flow easier. Increasing it tends toward stasis and thrombosis. Viscosity of blood depends largely upon changes in the number and volume of red blood corpuscles. It is increased in thromboangiitis obliterans by virtue of the marked increase in the volume of red blood corpuscles (lecithin content). With an increased coagulability and viscosity in the presence of hemolytic agents such as "tox lecithides" resulting in the liberation of fibrin ferment, a tendency to thrombus formation is established even without injury to the vessel wall.

CAPILLARY STASIS DUE TO PARALYSIS OF THE CAPILLARIOMOTOR MECHANISM

In thromboangiitis obliterans, the large and small vessels of the extremity present pathognomonic architectural lesions, such as thrombus formation, organization and canalization. The disturbance in the capillaries of the affected part does not present such a bizarre picture, though the end-results are more far reaching and deleterious. Very early in the disease rubor and pallor are noted by the patient. In fact, the changes in the color of the toes from pallor to a very deep pink and even purple and vice versa simulates Raynaud's disease so closely that only the absence of pulsations in the dorsal artery of the foot and the posterior tibial artery differentiates the one from the other.

Table II

MALES UNDER THE AGE OF THIRTY FREE FROM EVIDENCES OF THROMBOANGIITIS OBLITERANS

Control Cases	Date	Whl. Bl.	Plasma	Urine Choline
Dr. A.....	4/13/31	10.5	8.0	negative
Dr. K.....	4/13/31	10.4	8.1	negative
Dr. G.....	4/14/31	10.0	8.5	negative
Dr. G.....	4/14/31	10.9	8.8	negative
S. H.....	4/16/31	8.0	5.0	negative
D. S.....	4/16/31	11.0	10.0	negative
Orderly.....	4/27/31	10.8	8.1	negative
Dr. G.....	8/19/31	11.1	8.0	negative
Dr. K.....	8/19/31	10.5	8.1	negative
H. K.....	11/30/31	10.6	8.0	negative
G. B.....	4/ 5/30	11.0	....	negative
S. B.....	6/ 3/30	11.0	....	negative
M. F.....	5/ 7/30	10.0	....	negative
B. D.....	7/17/30	8.6	....	negative
H. R.....	1/22/31	10.0	8.8	negative
M. F.....	4/11/31	11.0	8.6	negative
Internes A....	11/30/31	11.0	8.2	negative
B....	11/30/31	10.8	8.6	negative
C....	11/30/31	11.2	8.5	negative
D....	11/30/31	11.0	8.4	negative
E....	12/12/31	11.8	8.5	negative
F.....	12/12/31	11.3	8.4	negative
G....	12/14/31	10.6	8.0	negative
H....	12/14/31	10.9	7.9	negative

These irritative capillary bed manifestations are seen early in the course of the disease and constitute the first stage in the capillary bed poisoning. In this stage, the capillaries retain their ability to contract but the peripheral circulation is characterized by its lack of tone and stability. States of extreme contraction may change abruptly into pronounced dilatation. As the disease progresses, the affected part takes on a fixed pinkish-purple discoloration (rubor). This is the paralytic or second stage in the capillary bed poisoning (Fig. 1). The capillaries are now devoid of their normal tone and no longer respond to stimuli with resulting contraction and relaxation, since the capillariomotor mechanism is destroyed. In this stage, the temperature of the affected part is always below normal and imparts a sensation of cold, both to examining hand and the patient, even though the skin presents a highly colored pinkish-purple hue commonly known as rubor.

The color of the skin depends mainly upon the state of dilatation of the cutaneous capillaries and not upon the rate of blood flow through them. The temperature of the skin, however, is determined primarily by the rate of blood flow, which in turn, depends upon the state of contraction or degree of obliteration of the lumen of the arteries or arterioles. A change in the color of the skin implies a change in the state of contraction or relaxation of its capillaries, but does not indicate anything about the state of the arteries or arterioles. In thromboangiitis obliterans, the tem-

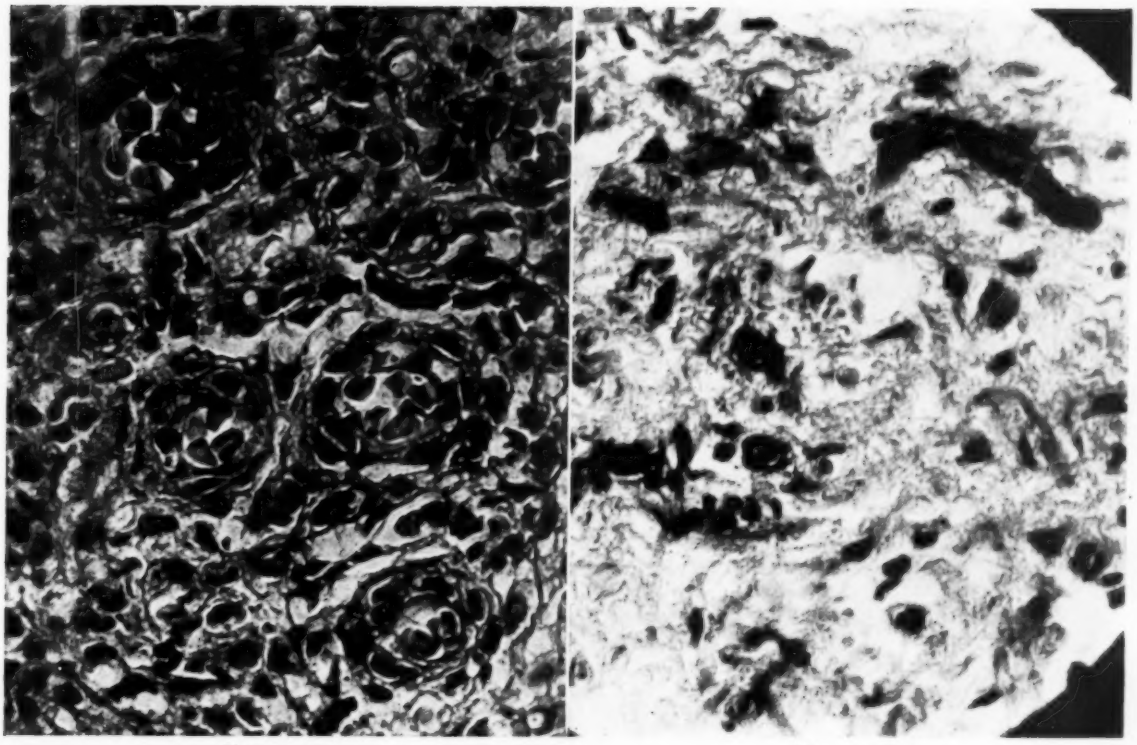

FIG. 1A. FIG. 1B.

FIG. 1. Second stage: paralysis of capillariomotor mechanism. A. J. S. Spec. area of rubor; capillaries markedly dilated and filled with blood cells. B. S. P. Spec. area of rubor; capillary dilatation, marked edema of interspaces.

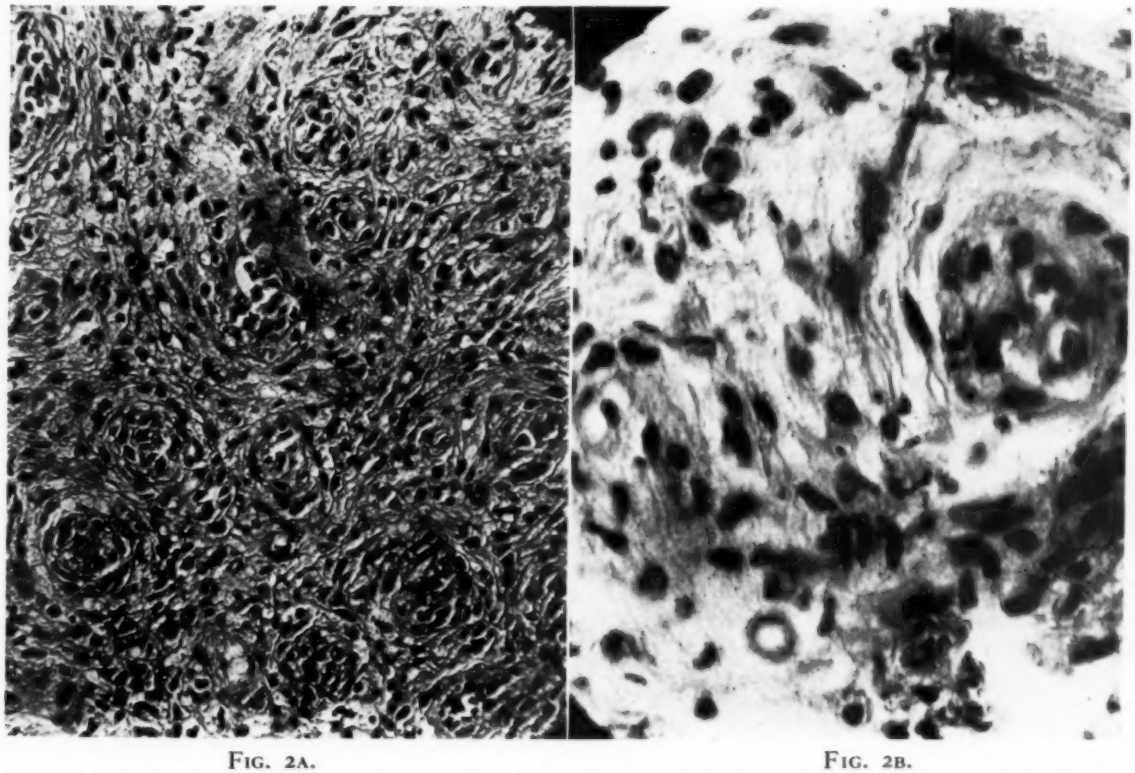

FIG. 2A. FIG. 2B.

FIG. 2. Third stage: capillary endothelial hyperplasia. A. J. S. Spec. gangrenous patch; marked endothelial hyperplasia with capillary closure. B. S. P. Spec. gangrenous patch; marked endothelial hyperplasia with complete closure of lumen.

perature of the part affected is lowered, as a result of the impediment to the rate of blood flow (thrombus formation), while the high color of the skin (rubor) is due to the resulting state of the paralyzed, dilated capillaries. In fact the venules and capillaries form a network of tubes filled with stagnant arteriovenous blood. On pressure over the discolored area, pallor appears, which on release is immediately replaced by the appearance of the previous pinkish-purple hue (rubor). On elevation of the extremity, the foot empties itself of the arteriovenous blood as though it were spilled out of a large-mouthed container, and takes on a deathly pallor interspersed here and there in the later stages by small violaceous islands of skin in which thrombotic closure of the capillaries has already taken place.

Fig. 3. J. K. Massive arteriosclerotic gangrene.

It is interesting to note that in thromboangiitis obliterans, gangrene seldom, if ever, affects a large area at one time, as one is apt to find in arteriosclerosis (Fig. 3). On the contrary, a minute area of necrosis appears on the toe in the form of an erosion, ulcer, or ecchymotic spot and slowly spreads until it involves the whole toe. At the same time or later, similar areas are affected in the remaining toes and similarly spread slowly towards the ankle (Fig. 4). The gangrene is not due to a large vessel thrombus proximal to the lesion, but rather to local capillary bed closure, as a result of stasis and thrombosis. In the advanced stages of the disease characterized by rapidly spreading gangrene, capillary endovasculitis is all too frequently observed to be ignored (Fig. 2). With this picture in mind one cannot help feeling that there are potent factors acting upon the capillary structures as well as the large and small vessels.

Fig. 4. S. P. Sequential local gangrene of toes.

EFFECT OF CHOLINE DERIVATIVES ON CAPILLARIES

In the decomposition of fats, large fatty acid molecules are broken down into

smaller ones, but probably are seldom, if ever, of pathological importance. It is quite otherwise with the products of decomposition of lecithin. From its molecule, choline is split off which in turn is easily oxidized into more highly poisonous compounds isomeric with muscarine, and by losing a molecule of water forms neurine, or may be converted into acetylcholine. If the split product of lecithin such as choline is involved in any way in the physiopathological processes of thromboangiitis obliterans, it is more probable that it is choline in the form of some much more active compound. There is considerable evidence that such compounds exist or may be formed from substances present in the body. Tavau, Hewit and Mange prepared a large number of choline derivatives, much more active than choline itself, which might conceivably be formed in the body. Reid-Hunt found a substance in the suprarenal glands, which had about fifty times the physiological activity of choline and which readily yielded choline as the result of simple chemical manipulations. One of the most active hemolytic agents known is "lysocithine," a choline ester formed from lecithin (Delezenne and Fourneau).

The urine normally is devoid of choline or at most contains but traces, and even after the feeding of 1 to 20 gm. per kilo by mouth to rats, none could be found (Shanks). Subcutaneous injections of choline do not especially increase the amount secreted in the urine. Evidently it is largely destroyed. In the face of these facts, it is interesting to note that in the reported series, choline often appeared in the urine most ostentatiously, while there was not a trace of it in the controls. An outstanding feature is the observation that wherein the blood showed a rather high lecithin value, i.e., 12.5 and above, the urine was usually negative to choline. On the other hand it was most often strongly positive at 11.5 or below normal. These findings seem to point to the fact that there is an attempt on the part of the organism to establish a normal lecithin threshhold. The increased katabolism of lecithin results in an increased amount of choline in the blood and its excretion in the urine. In view of the enormous toxicity of choline derivatives, there must always be considered the probability that such toxic compounds may be retained in the blood as a result of disturbed lecithin katabolism. With this in mind, experiments* were undertaken to ascertain the immediate and remote physiopathological effects of acetylcholine on the capillaries of a cock's comb. The results proved conclusively that after a short interval of vasomotor irritability, the comb took on a permanent diffuse rubor actually bordering upon asphyxia. Injection of acecholine solution no longer brought about a dilatory effect. The capillariomotor mechanism seemed to be completely paralyzed. Microscopic section (Fig. 5) showed extreme capillary

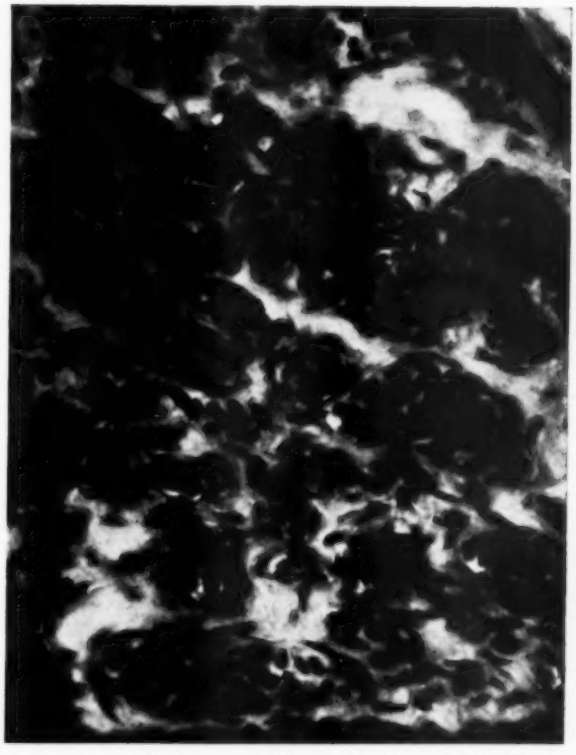

Fig. 5. Second stage; cock's comb No. 2. Capillaries strongly dilated, filled with closely packed corpuscles.

* Protocol on experiments, including technique and data, will appear in print at a later date.

dilatation, the corpuscles actually obstructing the lumen as a result of marked stasis. The counterpart of the foregoing picture is noted in the pregangrenous or second stage of thromboangiitis obliterans (Fig. 1).

RELATION OF EDEMA TO CAPILLARY STASIS

Edema of the foot and leg sets in late in the course of thromboangiitis obliterans and is dependent upon capillary permeability which in turn is normally increased by dilatation. It is therefore more or less dependent upon the state of the capillaries and their pressure relations.

The hydrostatic pressure of the blood in the capillaries determines, by its relation to the osmotic pressure of those colloids for which the capillary wall is impermeable, the direction and rate of exchange of water (isotonic salt solution) between the tissue spaces and the blood. When the capillary blood pressure is higher than the osmotic pressure, an excess of water over that attracted osmotically will filter through the capillary walls and edema will develop. When the capillary pressure is lower than the osmotic pressure, any excess of fluid in the tissue spaces will become absorbed, as can be observed to take place in most normal tissues. In thromboangiitis obliterans the capillary pressure is markedly increased on account of the extreme stasis in the capillaries as previously described. Edema develops and is persistent until the area which has undergone capillary stasis is completely eradicated.

Moreover, capillary pressure in the foot is dependent upon the "venous pump" (Hooker, 1911). The pumping action is due to compression of veins by muscular movements. Any compression of a venous segment will partially empty that segment in a central direction, by which action the pressure is lowered and the segment refilled. In walking (Krough) the pressure in the veins of the foot is usually reduced to near zero, the capillary pressure in the foot becomes higher than normal osmotic or effective pressure of the blood, and filtration edema appears. In thrombo angiitis obliterans the contractions of the voluntary muscles of the affected limb in the late stages are at a minimum and the "venous pump" becomes a negligible factor. The capillary pressure is increased accordingly and filtra tion edema of thefoot and leg develops.

MODUS OPERANDI OF CLAUDICATION

In order to appreciate the modus operandi of claudication in thromboangiitis obliterans, it is necessary to review the physiology of muscle contraction. In a long series of researches, Embden, Meyerhoff and their associates have established an intimate correlation between the conversion of carbohydrates into lactic acid in the contracting muscle, and the appearance of phosphates in the latter. The phosphoric acid radicle in the lecithin becomes highly important when we realize the role it plays in the intermediary metabolism of the carbohydrates, for a considerable portion of the phosphoric acid released from the phosphatides (lecithin) goes to form hexose or similar phosphates.

According to present-day ideas the succession of events in muscle contraction as far as they concern carbohydrates are as follows: the muscle glycogen combines with phosphate (presumably passing through some intermediate simpler form of carbohydrate), the resulting hexose phosphate is broken down by the muscle enzyme or enzymes with the liberation of phosphoric acid and lactic acid and it is during this phase of acid production that the muscle fibers contract.

The whole contractile phase, which includes the onset and maintenance of contraction, and the relaxation to the normal resting condition, proceeds with a relatively small heat output and can take place in the entire absence of oxygen. It is therefore called the "anaerobic phase." During the recovery or "aerobic phase" which follows the relaxation and which is only completed in the presence of oxygen, there is a prolonged heat production. The uptake of oxygen during this phase corre-

sponds to the oxidative removal of the lactic acid formed during the first phase and its reconversion into glycogen. In the absence of an adequate supply of oxygen (impaired circulation) during the second phase of recovery, the lactic acid formed during the first phase accumulates in the muscle tissue, which becomes fatigued or contracts with less vigor or not at all.

Furthermore, Meyerhoff found that the transfer of oxygen to dried muscle in the presence of a sulphydryl compound ceased under the conditions of his experiment, if the muscle were thoroughly extracted with alcohol and ether, thus depriving the muscle of its phosphatides (lecithin). Any disturbance in the phosphatide content of muscle tissues would therefore seriously interfere with the oxygen uptake of the muscle, so necessary to the retransformation of lactic acid through hexose phosphate and glucose to glycogen. In thromboangiitis obliterans, the phosphatides (lecithin) of the tissues are mobilized and split up into choline and its derivatives as shown in the early part of the paper (Fig. 1). A deficiency in the oxygen available for the tissues, whether due to the decreased oxygen supply of impaired circulation (thrombosis of vessels), or to a decrease in the oxygen uptake of the muscles, results in the endogenous production of lactic acid and its retention, with resulting claudication.

EFFECT OF LECITHIN ON SENSORY PATHS

The pain associated with the pregangrenous and gangrenous stages of this disease is most excruciating and intractible. In fact, it is one of the few diseases in which the average doses of the commonly used narcotics do not exert their usual pharmacological action on the sensory apparatus. The patient describes his pain in the most bizarre fashion. Mere exposure of the affected part to the air is sufficient to cause pain. While the patient may experience an actual freezing sensation of the affected foot, application of heat in any form is most unbearable. The pain in thromboangiitis obliterans finds its counterpart in that of causalgia, a most violent form of nerve irritation described by Weir, Mitchell, Morehouse and Keene in 1864. In thromboangiitis obliterans, the myelin of the nerves, which is rich in lecithin, has been shown to undergo almost complete absorption (Brown, Allen and Mahorner). This finding, coupled with the fact that choline is abundantly found in the urine in thromboangiitis obliterans, makes it quite possible that the impoverished lecithin content of the nerves has a direct bearing on the irritability of the sensory fibers in the part affected. In this connection it is rather interesting to note that mounding, a form of muscle irritability, has been elicited in all the cases examined.

Smoking, no doubt, aggravates the pain in thromboangiitis obliterans. Nicotine, however, is not the active agent producing the deleterious effect. Pyridine, a fat-soluble organic basic substance is a normal constituent of tobacco and may possibly change the physical structure of the lecithin in the tissues.

TREATMENT

The principles carried out in the treatment of thromboangiitis obliterans are based on an attempt to regulate the lecithin intake and the neutralization of the poisonous effects of choline and its derivatives. In many ways the action of cholesterol appears to be definitely antagonistic to that of lecithin, in that a certain balance of these substances may be a necessary part of the cell mechanism. The antagonistic effect of cholesterol to the hemolysing action of "lysolecithin" is a good example. It is very probable that in the tissues and blood, combinations such as that of cholesterol with "lysolecithin" may occur. The highly developed cholesterol-regulating mechanism in the female may have an important bearing upon the low incidence of thromboangiitis obliterans in that sex.

In the first half of the series, cholesterol injected intramuscularly seemed to have a most favorable influence upon the symp-

toms, though the thrombotic tendency was unimproved and at times enhanced, probably because of the increase in the viscosity

paralysis of the capillariomotor mechanism was very evident and of long duration, the rubor was apparently not affected. While

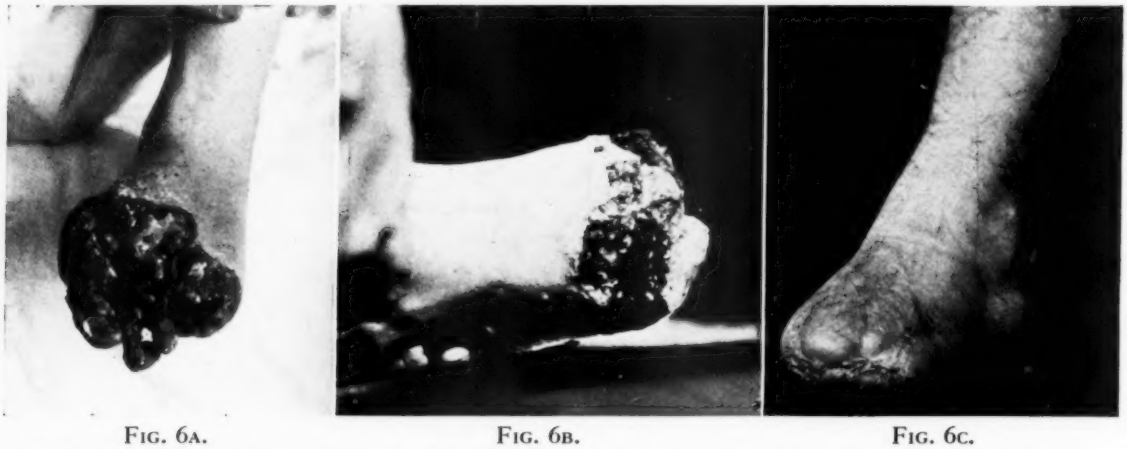

FIG. 6A. FIG. 6B. FIG. 6C.

FIG. 6. Rapidly advancing gangrenous lesions. A. All toes gangrenous, process extending up to metatarsophalangeal joint. B. Gangrenous areas completely cast off. C. Healed; good functioning stump at metatarsophalangeal joint.

and coagulability of the blood. It was very apparent that a substance that would convert the degradation products of lecithin into harmless compounds and have no effect upon the composition of the blood would be preferable. After much experimenting, it was found that choline and its derivatives are easily converted by activated sulphur into non-toxic compounds. In the latter half of the series activated sulphur was injected intravenously every other day for the first three weeks and twice a week thereafter for two months. All foods rich in lecithin such as fish, liver, eggs and meats, were removed from the diet.

Under this regime, pain was the first symptom to disappear. Patients who have been accustomed to spend sleepless nights in a chair, have enjoyed the comforts of a bed after three weeks of treatment. Trophic ulcers and gangrenous areas were replaced with healthy granulation tissue, followed by rapid healing. The usual pale and worn-out facies vanished. An outstanding feature indicative of improvement was the progressive gain in weight and the noticeable growth of the nails. In the early cases, the rubor was gradually substituted by the normal color of the part. Where claudication was practically the first episode calling attention to the disease, this symptom was the most stubborn to control and the last to disappear.

When seen in the advanced gangrenous stages, notwithstanding the pathological lesions, more than could be hoped for was obtained. Pain was controlled immediately and the extremities preserved with the loss of the least amount of tissue. (Fig. 6.) In the reported series of 50 cases, 3 were of the fulminating, rapidly advancing gangrenous types, all young men under thirty. In the face of the vast extent of the process and its spread up the dorsum and plantar surface of the foot, they were spared a high amputation and now enjoy good functioning stumps at the metatarsophalangeal junction. In the cases in which arteriosclerotic changes were evident (thromboangiitis obliterans often complicates the former) the prognosis was very guarded. Four cases in this series showed arteriosclerotic changes and in all high amputations were performed.

SUMMARY

The mobilization of lecithin from the tissues into the blood stream with the production and retention of degredation

products such as choline and its derivatives may well explain the thrombotic tendency, capillary bed disturbance, claudication and intractable pain in thromboangiitis obliterans. The capillaries are involved in the pathological process as well as the large and small vessels. The capillary lesions may be divided into three stages: (1) the irritative and vasomotor disturbances; (2) paralysis of the capillariomotor mechanism with marked dilatation of the capillaries resulting in rubor and stasis; (3) pregangrenous or gangrenous stage, in which there is capillary thrombosis and endovasculitis. Treatment executed on the basis of the limitation of lecithin intake and the detoxication of choline derivatives by the intravenous use of activated sulphur gave most promising and lasting results.

I wish to acknowledge my great indebtedness to Drs. Mendel Jacobi and Herman Bolker, in charge of the Pathological Departments of the Beth Moses and the Beth El Hospitals, for their painstaking labor in the preparation and interpretation of the material entrusted to them. My thanks is likewise due to Dr. I. Charles Zuckerman for the admirable manner and the meticulous character in which the blood lecithin values have been ascertained.

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[For Remainder of References see p. 288.]

OVULATION, MENSTRUATION AND THE HORMONES *

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IN considering the effect of the hormones on the uterus and ovaries, it seems advisable to summarize a few facts regarding the synchronous ovarian and uterine cycles and menstrual blood before considering the details of clinical results.

The graafian follicle begins to mature following menstruation and at maturity the follicle ruptures, extruding the ovum into the peritoneal cavity. This is apt to occur at about the fourteenth day following the onset of menstruation. The developing follicle and its successor, the corpus luteum, produce the female sex homone which is responsible for the rebuilding of the dismantled endometrium. The corpus luteum also produces simultaneously the nidatory hormone, progestin. The uterus is thus prepared for the reception of a fertilized ovum. The span of life of the unfertilized ovum is not definitely known. Allen and Pratt have shown that it is probably no longer than six days. If the ovum is not fertilized it probably dies on the twenty-eighth day and the corpus luteum degenerates and gradual involution takes place over the following four weeks.

If the ovum is fertilized, then the corpus luteum grows larger instead of degenerating, and the effect of progestin on the endometrium persists; the endometrium becomes the decidua and retains in a more definite way its premenstrual secretory function and appearance.

During the follicle growth characteristic changes occur in the endometrium and the typical secretory phase appears as a result of the corpus luteum hormone, progestin, during the last two weeks of the cycle.

It is authoritatively claimed that during the thirty to thirty-five years of sexual life of the average woman, only 360 to 420 follicles mature completely and rupture.

ATRESIA OF THE FOLLICLE AND RETENTION CYSTS

Some follicles develop to a degree and perish by the process of atresia; others develop and persist. Consequently the law of averages would show that 299 out of 300 primordial cells terminate in atretic bodies.

NORMAL ENDOMETRIAL CYCLE

The uterine mucosa has normally such definite stages of growth, development and disintegration during the twenty-eight day cycle that the clinician and pathologist must be thoroughly familiar with the microscopical findings. Six days after menstruation the glandular cells are high columnar with the stromal cells very close together. Thirteen days after menses the epithelial cells are still high columnar and show pseudo-stratification with an edematous stroma and cells far apart. About twenty-two days after menses the epithelial cells become low columnar cuboidal in type and the stroma cells are larger and closer together. Twenty-nine days after menstruation the epithelial cells show degeneration, and marked edema of stroma is evident.

MORPHOLOGY OF MENSTRUAL BLOOD

The morphology of the menstrual blood has a diagnostic value clinically, and in about 90 per cent of the cases true menstrual discharge can be identified. Therefore it becomes of value in determining the difference between normal uterine bleeding, abnormal uterine bleeding and bleeding from other vaginal sources.

Uterine desquamation seems to occur before the first day of menstrual bleeding, is greatest on the second day, and almost disappears on the fourth. Uterine stromal

* Read before the Sacramento Society for Medical Improvement, May 17, 1932.

cells appear in greater percentages on the second and third days and also are only rarely found on the fourth. Vaginal spindles seem to be present in almost all cases throughout, though the vaginal plaques become less each day. When uterine elements are found in the blood it is evidently of menstrual origin.

Geist of New York presents the following table:

PERCENTAGE VARIATION OF UTERINE VAGINAL ELEMENTS ON SUCCESSIVE MENSTRUAL DAYS

	1st Day Per Cent	2nd Day Per Cent	3rd Day Per Cent	4th Day Per Cent
Uterine epithelium.....	50	74	25	3
Uterine stroma.....	75	90	91	4
Vaginal spindles.....	95	95	82	100
Vaginal plaques.....	90	72	55	25

Most investigators, according to Graves, have come to the conclusion that "menstruation is not the homologue of proestrum, preparatory to ovulation, but on the contrary is a late result of ovulation and represents a disintegration of a structure that has been built up, but not required, for the reception of the fertilized egg," although Aschner believes that the flow represents an excretion following metabolic changes. Whether the uterine bleeding is a positive or negative factor, it is due to an active substance originating outside of the ovaries now proved to be in the anterior pituitary.

Corner's experiments with macacus rhesus monkeys have proved that menstruation can take place without ovulation, and it is considered by Graves and others including Allen that atresia can at times supply sufficient follicular hormone to bring on a premenstrual condition which is followed by menstrual flow.

Novak believes the follicle to be responsible for the early slow hypertrophic changes in the endometrium up to the beginning of the secretory phase; and the premenstrual phase is associated with the development and maturation of the corpus luteum. Corner has definitely established the existence of a special corpus luteum hormone. Zondek and Ascheim designate the pituitary as the "regulator of menstrual rhythm." There are some who now consider the anterior pituitary to have five different hormones. Production of menstruation requires then the hormones of the follicle, corpus luteum and anterior pituitary.

OVULATION

Meyer, Ruge, Schroeder and Corner have all come to the conclusion that rupture of the graafian follicle occurs about fourteen to sixteen days after the onset of menstruation. And because the development of the corpus luteum is followed by changes in the endometrium there is doubtless a hormonal factor causing these premenstrual changes. The time relationship between ovulation and menstruation is rapidly becoming better understood but the true relationship between these two and conception is still a very difficult problem.

ENDOMETRIAL HYPERPLASIA

In the endometrial hyperplasias the actively growing gland cells are heaped together in a pseudo-stratified manner showing numerous mitotic figures and the stromal cells are small but closer together than those thirteen or fourteen days after menses in a normal patient.

In a case of endometrial hyperplasia there is such a predominance of these retention cysts that the follicular hormone effect causes a persistent growth of the endometrium with the irregular hypertrophy so often seen by the pathologist, or macroscopically by the surgeon and in the hysterogram by the roentgenologist. In the absence of the lutein hormone, progestin, the secretory phase of the endometrium does not occur. There is some discussion and difference of opinion regarding the etiology of the persistent bleeding, and the histopathological condition of the endometrium. The dysplasia of the

endometrium results in localized thrombosis and necrosis with consequent hemorrhage. The persistent ovarian follicle at times, instead of becoming atretic, develops into a cystic condition, and whether the endometrial hyperplasia is the effect of this cystic fluid hormone or the anterior pituitary, or both, on the endometrium is being rapidly determined by a number of investigators.

Cystic fluid removed from an ovary with no corpus luteum and injected into a female laboratory animal is followed by the same uterine changes of endometrial hyperplasia in the animal as were exhibited in the patient. These patients are evidently suffering from a lack of corpus luteum hormone. This one fact is surely the keynote in the clinical treatment of such cases.

ISOLATION OF OVARIAN HORMONES

The effort to isolate the ovarian hormones has been persistently going on for a number of years, mainly because ovarian gland extract therapy has been unsatisfactory. The isolation of the follicular hormone in a crystalline form, a colorless white crystal, was accomplished by Allen and Doisy in 1928, and a few months later it was also isolated by Butenandt of Germany. This is a triple unsaturated oxyketone with the formula $C_{18}H_{22}O_2$. Theelol has the formula $C_{18}H_{24}O_3$ and is about one-fifth as potent as that of $C_{18}H_{22}O_2$.

This has stimulated an active interest again in clinical therapy relative to the disorders of menstruation. But, because many were still unsatisfied, a continued research has developed many other factors including the role of the anterior pituitary.

Follicular hormone is found in very large quantities in the placenta, the blood and urine of pregnant woman. It is from this latter source that most of the products now available are obtained. It takes in the neighborhood of 1000 gal. of urine to obtain 1 gm. of follicular hormone, which is about 8,000,000 mouse units.

The fact that the corpus luteum develops a hormone has been well established, the final scientific proofs being presented by Corner in 1929. The dual secretion of the ovary also has now been well established. The hormone of the follicle and that of the corpus luteum are positively antagonistic, as has been demonstrated in many ways experimentally and is easily proved clinically.

CLINICAL EXPERIENCE

The laboratory research must be followed by an effort clinically to evaluate the practical use of the various hormone preparations in an effort to relieve the distress and discomfort of those whose endocrine systems do not seem to be properly geared, or whose gears are not properly meshing, or, as most investigators prefer to consider it, whose body chemistry is disturbed by an unbalanced percentage of the various hormones. During the past two years our work has been with the use of the follicular hormone and the luteinizing anterior pituitary; we have treated our cases with these products exclusively, not in a spirit of over-enthusiasm but in order to determine the effect statistically of each preparation.

Because so many of the symptoms of menstrual disorders are of the subjective character we have found much more satisfaction in drawing conclusions in the treatment of private patients than of the clinic types who are financially and socially handicapped.

We have worked with cases of amenorrhea, menorrhagia, metrorrhagia, dysmenorrhea, oligomenorrhea, and the conditions attendant to the menopause.

Amenorrhea. Primary amenorrhea cases have exhibited an improvement in general but the establishment of regular menstruation has not been obtained. At times one becomes enthusiastic only to find that in another month or two black disappointment is encountered. The definite tonic effect on the individual with the administration of follicular hormone is nevertheless obvious in every case. In secondary amenorrhea our results were

quite gratifying, and in the primary irregularities administration of the follicular hormone has been universally satisfactory.

There are, in my opinion, patients with relative deficiency of follicular hormone due to a loss of so much of the normal amount formed in the ovaries due to "kidney permeability" that they are apt to develop a secondary amenorrhea. Massive doses of follicular hormone are required to bring about the desired results in such cases, but the response is much more rapid than in those whose ovarian function is impaired. Patients who have had varying degrees of partial castration are very grateful when given the proper individual doses of follicular hormone with the required regularity.

Functional menorrhagia and metrorrhagia of early years of menstrual life show clinically an endometrial hyperplasia and, in some cases, excess of female sex hormone in blood and urine. These cases, in which there is a relative or real excess of follicular hormone and very little if any corpus luteum hormone, progestin, obviously are not proper cases in which to administer follicular sex hormone. But the luteinizing pre-pituitary hormone does give excellent results in these young girls just as well as in the hyperplasias of older women nearing the menopause. Apparently in these hyperplasias in which bleeding is more or less slow this "prepituitary B" will prevent the necessity of roentgen ray or radium therapy or surgery. But in the severe hemorrhages it does not seem to have sufficiently rapid effect.

G. V. S. Smith and O. W. Smith have delved into the functions of progestin, the hormone of the corpus luteum, in the menstrual cycle and in pregnancy. Among other findings they observed that it also has an inhibitory action on the function of follicle hormone, the continued and unantagonized action of which is responsible for functional uterine bleeding. As potent products of progestin are not available for therapeutic use, the prepituitary B hormone obtained from pregnancy urine is employed to evoke luteinization and consequent production of progestin to control the harmful effect of the follicular hormone on the endometrium.

Alvarez classifies a certain group of people as "constitutional inadequates." These naturally have a poor background, a poor foundation. There are those who do not develop genitally to maturity and others who are just on the threshold and manifest their "inadequacy" by only two or three menstrual pads monthly. Some have a few of the angioneurotic symptoms frequently found in menopausal women. Most clinicians report a comparative failure in these patients by the use of follicular hormone. In our experience both of these groups have shown most pleasing improvement under treatment with this product. In fact these cases seem to offer the most gratifying results and it seems to me to be really constructive therapy. Just how long they may be "carried over" is problematical. But one naturally hopes for a satisfactory "prolan A" preparation that will stimulate the ovaries of these patients to renewed function.

The series of tests recently described by R. T. Frank and now easily performed in small laboratories include:

1. Female sex hormone test of the blood.
2. Female sex hormone test of the urine.
3. Prepituitary maturity factor test of the blood.
4. Prepituitary maturity factor test of the urine.

The prepituitary tests are primarily simplified methods of the work done originally by Aschheim and Zondek. Just as soon as these become laboratory methods the clinician will have less of the "trial and error" methods to follow in his administration of the ovarian and prepituitary hormones. At such a time there will be fewer "negative" results clinically and a greater percentage of satisfactory reports in each series.

Dysmenorrhea. It has been reported that in many cases of dysmenorrhea the administration of the follicular hormone

seemed helpful, and in a small percentage of cases apparently curative. Rather surprising results are obtained in the severe cramps of young girls, or at least in the majority of them. The belief that many of these are due to a lack of sufficient vascularization of the uterus naturally has prompted us to use various methods of causing pelvic congestion. We were satisfied many months ago that the follicle hormone not only does that but increases the congestion of all of the female genital organs, so we felt thoroughly justified in the use of that hormone in such patients, and now believe sincerely in its efficiency in such types of dysmenorrhea. In some of my cases, what has seemed an overdose apparently increased the cramps. It would require a full discussion of dysmenorrhea to determine logically the place of the hormones in this distressing phase of the early years of menstruation in some women. Most of these patients are permanently cured of dysmenorrhea by pregnancy and normal delivery. The hormonal changes during pregnancy cannot be determined by a clinician. Such work must be continued by Fluhman, Novak, Corner, Zondek and hundreds of others who are doing the real research work now.

The physician does not enjoy the empirical use of any form of therapy. It therefore becomes necessary for the clinician to learn the whys and wherefores. As far as I know there are many questions of prime importance still unanswered. The follicular hormone is commercially obtained from the urine of pregnant women. It is obvious then that there is not only a larger amount of follicular hormone developed in the body during pregnancy, but there is a very large amount excreted, not needed, possibly wasted as far as that individual is concerned. Is it possible that this is all formed in the ovaries, or is the placenta not only a place where the follicular hormone is stored in large quantities but also the source of the hormone? Doubtless Collip can answer this question after his experience with emmenin.

Menopause. During the menopause, when there is a gradual reduction of ovarian function, theoretically one might expect relief of many of the subjective symptoms complained of by the judicious administration of both ovarian hormones. Actually in practice almost all of the commercial preparations seem to have some beneficial action. Obviously there would be no justification for the administration of the prepituitary hormone in the frank menopause. Naturally the effort of the physician is to relieve the angioneurotic symptoms during these distressing months. Replacement therapy with the follicular hormone administered in various ways to a large number of patients justifies our belief in the efficacy of the product. They seem better generally and claim to be greatly improved. The follicular sex hormone preparation has apparently made them feel better than others.

Uterine Bleeding. The anterior pituitary lobe undergoes marked hypertrophy during gestation, and Novak places this as the cause of the acromegaloid changes seen at times in late pregnancies.

According to Schroeder the cause of bleeding in the hyperplasias is due to small areas of necrosis, thrombosis or necrobiosis. But it is generally considered to be some change in the permeability of the blood vessels. The endometrial lesion itself cannot account for it because the same histological picture may be found with amenorrhea.

HORMONAL THERAPY: DOSAGE AND MODE OF ADMINISTRATION

In the administration of the follicular hormone one should know the amounts needed, the minimum and maximum doses and how to offset the effects of incorrect amounts in the individual. How much is absorbed when it is given intramuscularly and orally? What percentage, if any, is changed chemically? Is there a saturation point varying in different individuals dependent upon their physiological needs? Is there a deleterious effect on the indi-

vidual by what might be termed an overdose? Why is one patient given complete relief with 150 R.U. intramuscularly while another whose subjective symptoms seem parallel is not relieved by a dose short of 900 to 1000 R.U. intramuscularly, if there is no need of individualizing treatment? What happens to the hormone, not excreted, after it leaves the blood stream? Is it stored up for a certain period of time, and if so, where? How soon is the effect of an individual dose noted by the patient and how long after the last of a series does the effect seem to be noted? It may be stated here that some of the most frequently subjective changes due to the administration of follicular sex hormone are relaxation from nervousness, disappearance of phobias, return of mental calm, relief of various headaches and cerebellar pressures, increase in ambition and enthusiasm and capacity for physical work or exercise. Fatigue is relieved and the face and eyes become brighter. A general increase in libido is noted by the majority of patients. We are arriving at the conclusion that follicular hormone has not only a replacement value but that it is a definite stimulant as well. And this may account for the fact that some report a sensation of well-being for about ten days after the last dose of a series followed by a gradual return of their predominating symptoms. The most frequent statement of results made by those who have been languid (or nervous) semi-invalids for months, is that they felt so good that they "got up and cleaned house."

Dosage of (follicular sex hormone progynon) ranges from 25 to 300 R.U. by hypodermic each month up to 600 R.U. by mouth daily for three weeks. I now have several patients taking 600 R.U. orally each day for three weeks out of every four. This gives them in the neighborhood of 12,000 R.U. monthly. Many have stated and some still believe that 50 or 100 R.U. per dose is insufficient to give clinical results, but it is the clinicians who will determine that. The majority of our cases are doing well on a total of 150 to 300 R.U. intramuscularly or from 300 to 3000 R.U. orally each month.

During the last six months we have used only two products in our clinical experiments, namely the follicular hormone (in the form of ampoules, or tablets of 30, 200 and 600 R.U. each) and the luteinizing hormone of the anterior pituitary as used by Novak. The dose of the early product of luteinizing prepituitary hormone seemed to be 50 to 100 R.U. daily. In our experience 50 R.U. were all that could be given in daily doses intramuscularly without causing a general reaction, but the later product permits the safe use of 200 R.U. daily.

For many years I have employed commercial preparations of ovarian tissue with uncertain results, but during the past two years the follicular hormone, which I have mentioned, has given me the best results clinically. Of course, the action of the luteinizing prepituitary hormone is remarkable in the type of cases reported by Novak. Naturally, I am extremely grateful to Drs. Stragnell and Sharpe for the generous quantities of material they have furnished me for clinical use.

During the last two years we have treated over 200 patients, mostly falling into the menopausal and secondary amenorrhea groups. Some of the most interesting cases are the so-called "neurotics." In almost every instance one can obtain a history of a greatly reduced menstrual flow. The results of the hormone therapy in these cases are only short of miraculous. You may perhaps suspect that the effect is purely psychological, but I am sincere in my claim that a large number of women of twenty-five to thirty-five become neurotic or hysterical because their ovaries are not properly functioning. So that a monthly replacement of ovarian hormones will correct their troubles until such time as the prolan A and prolan B are both available commercially. These younger women, I believe, can have their ovarian system stimulated to function properly. But those of forty-five to fifty-eight years old are

in need only of the required monthly replacement doses to permit them to enter gracefully the period past the so-called "change of life." Obviously the pre-pituitary hormones are not indicated in the latter.

Scanty menses in young women and at the climacteric are accompanied by angioneurotic dyscrasias, obesity, susceptibility to infections, etc., and one-third of hypomenorrheic patients show arthritic, rheumatic or neuralgic conditions. Pains in this latter group are worse before and better after menstruation. Relief is obtained by treatment with ovarian hormone.

Under the care of Molle, 2½ to 3 pound premature infants improved in development by the daily use of follicular hormone.

So-called "menstrual migraine" in some cases is reduced in severity by the administration of corpus luteum injections daily for ten days pre-menstrual. These patients include those who have an actual reduction of corpus luteum hormone or a relative increase of follicular hormone.

Basal headaches occurring during the menopause are promptly relieved by correct doses of follicular sex hormone.

Theoretically, the luteinizing pre-pituitary hormone should be of value in threatened abortion, and helpful in preventing premature delivery as large doses of follicular hormone are considered abortifacient.

In the use of these newer chemicals the over-enthusiastic clinician is apt to make claims that the test of time and a larger series of cases prove to have been unwarranted. But over a period of two years we have watched a sufficiently large group to be able to justify our claims that the follicular hormone in the form of progynon, and Novak's luteinizing pre-pituitary are very helpful agents in the treatment of many menstrual disturbances.

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*Continued from p. 226.

THE MANAGEMENT OF
ABRUPTIO PLACENTAE

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THIS pathologic condition is variously known as abruptio placentae, premature separation of the placenta, accidental hemorrhage, ablatio placentae and when it is associated with hemorrhage between the uterine muscle fibers, as uteroplacental apoplexy.

Holmes places the incidence of abruptio as 1 in 200 cases. In Polak's series it occurred once in 305 cases. Polak's figures represent only those cases which were diagnosed as abruptio placentae before or during labor, and required treatment. They do not include many cases in which the separation was so slight that no clinical significance was attached to the vaginal bleeding. Goethals in the Boston Lying-In Hospital encountered this complication once in 94 deliveries.

This condition is frequently found in association with low implantation of the placenta. The case is then characterized by the signs and symptoms of both conditions.

Polak in his last article on this subject gives as the predisposing causes, toxemia, torsion of the uterus, and endometrial disease. In Holme's collection of 200 cases from the literature, accident was given as the cause in 67. Williams, in the last edition of his textbook (1930) stated that, in his opinion, the influence of trauma as a cause of premature separation of the placenta is exaggerated. Chronic nephritis was present only twice in 57 of his cases, though albuminuria was present in 33 of the 57. He thought that albuminuria is an accidental complication or a manifestation of an acute toxemic process. Hofbauer states that the preponderance of evidence accumulated during the last few years, both from clinical observation and autopsy findings, points to a toxic element as the underlying factor in the production of abruptio placentae. Kellogg and Young have called attention to the marked toxic symptoms which are present in many cases of abruptio placentae, and Young has attributed these symptoms to red infarcts and blood resorption. Brown showed that in animals in whom a chronic nephritis has been caused by a previous injection of sodium oxalate, abruptio placentae can be induced by the injection of uranium nitrate followed by an injection of B. pyocyaneus. In the absence of chronic nephritis, injections of B. pyocyaneus are usually not sufficient to cause hemorrhage even though the injection of the organisms has caused an acute nephritis. In all the animals suffering from experimentally produced chronic nephritis, the liver function test was normal. O'Connor reports 37 cases, in 33 of which toxemia was present.

In Goethal's series 24.2 per cent showed a toxemic background, and 4.7 per cent occurred in association with eclampsia. During eleven years, abruptio placentae occurred four times more frequently in toxemic patients than in the general series of deliveries.

In my series of 15 cases, toxemia was present 12 times. Eclampsia was present once.

Hofbauer was able to produce identical lesions of abruptio by injections of histamine.

DeLee in 1901 drew attention to the hemophilic nature of some of these cases, and Williams compared the pathologic changes in the uterus to the local manifestations of certain venomous snake bites.

According to DeLee, studies of the uterus, endometrium and placenta have

not yielded much of value. The only constant local finding has been a more or less marked bloody infiltration of the decidua and myometrium. This, he surmises, is due to the same noxa which causes an abnormal permeability of the spongiosa blood vessels. Whether a diseased ovum furnishes the noxa or the latter reaches the uterus from elsewhere is not known, but the evidence points to a placental toxin. The role that infection plays needs study. In the presence of one of the foregoing conditions even a slight trauma may cause a separation of the placenta. A hemorrhage having once been started, blood works its way between the soft layers of the decidua and completes the abruption. A jar, coitus, turning suddenly in bed, a blow or kick in the abdomen, the action of a purge, severe coughing, tension of a short umbilical cord, and profound mental emotions have all been assigned as causes of abruptio placentae.

In my series of 15 cases the causes were: toxemia of pregnancy, 12; infection, 1; profound mental emotion, 1; unknown, 1.

One of the toxemic patients gave a history of an automobile accident two weeks before delivery.

The parity of the patients in my series was as follows: para 0, 7; para i, 5; para ii, 1; para iii, 1; unknown, 1.

In Goethal's series 39 were primiparae and 89 multiparae.

Uteroplacental apoplexy occurred five times in my series. According to Hofbauer the condition is due to a lesion of the walls of the capillaries and small veins, which allows plasma and red blood cells to leave the vessels in the affected areas, thus giving rise to marked edema and hemorrhages by which the muscle bundles are spread apart. It is the capillary toxicosis which constitutes the prominent feature of this condition and of eclampsia as well. It is the presence of platelet thrombi and endothelial injury in the uterine wall, and in the adjacent tissues, which characterizes the peculiar pathology of abruptio placentae.

This condition is always accompanied by a marked hemorrhage, unless the fetus has been dead long enough to allow thrombosis to occur in the uterine sinuses. Since the full uterus cannot retract over its contents, the sinuses remain open and the cavity of the uterus is exposed to the full force of the arterial circulation. The pathologic sequence according to Williams is an effusion of blood into the decidua basalis. The decidua then splits and a placental hematoma develops, which leads to separation, compression and ultimate deprivation of function of the placenta attached to it. The uterus being full, it cannot contract down on the blood vessels and the latter therefore remain open and bleed.

The hemorrhage is either concealed or apparent. It may remain concealed under the following conditions: (1) when there is an effusion of blood behind the placenta, its margins still remaining adherent; (2) when the placenta is completely separated while the membranes retain their attachment to the uterine wall; (3) blood gaining access to the amniotic cavity after breaking through the membranes and the amniotic sac remains intact below; and (4) the head is so accurately applied to the lower uterine segment that the blood cannot make its way past it.

Uteroplacental apoplexy is a term introduced by Couvelaire to describe the appearance of the uterus in some cases of abruptio placentae. The uterus in this condition looks like a large ovarian cyst whose pedicle has become twisted. The uterus looks distinctly blue and in one of my cases the anesthetist on seeing the uterus exclaimed in horror, "Its gangrenous." That particular patient made an excellent recovery, and I have delivered her since of a normal living infant by cesarean section.

The symptoms as a rule depend on the severity of the bleeding and the amount of shock. Most authors classify the cases as mild, moderately severe and severe. Mild cases may manifest themselves by a small amount of external bleeding, some moder-

ate pain over the uterus and a slight degree of shock, accompanied by a more or less board-like uterus which is slightly or moderately tender. In some cases the uterus has been found to be boggy. The blood pressure is usually elevated, especially if it is recorded prior to the onset of symptoms. The urine usually contains albumin. In the severe cases all the signs and symptoms are more marked. There is usually a sudden onset with very severe abdominal pain, a profound degree of shock, a semicomatose or even a completely comatose state and an extremely tender and hard, ligneous, board-like uterus. The pain is usually constant though it may be cramp-like, colicky or bearing down. In cases where there is concealed hemorrhage the uterus will be found to be perceptibly increasing in size. There is as a rule a fall in the systolic pressure and a marked change for the worse in the character of the pulse. There is frequently found an area of uterine surface which is more tender than the rest of the uterus. In this excessively tender area, especially in the concealed hemorrhage cases, a boss forms on the uterine surface, causing asymmetry. This area corresponds to the site of the retroplacental blood collection. There is a diminution in red blood cells and in the hemoglobin content of the blood and the extent of this decrease should be determined by hourly blood examinations.

In the mild cases, the fetal heart can usually be heard and a living fetus is frequently delivered. In the moderately severe and in the grave cases, the fetal heart as a rule cannot be heard and the infants are most frequently stillborn.

Labor usually commences with the onset of symptoms.

Abruption may occur during labor. A gush of blood and clots follow the birth of the child, confirming the diagnosis.

Postpartum hemorrhage is a frequent complication. At times the bleeding assumes a hemophilic character, there being a continuous oozing of blood of a watery consistency.

The prognosis is always serious. According to DeLee, it is safe to say that one-half of the women and 95 per cent of the babies in complete detachments with concealed hemorrhage will die, while a larger proportion will be saved with partial detachment under skillful treatment. Goodell found 54 maternal deaths in 106 grave cases. Greenhill reports 3 maternal deaths in 82 cases, Burgess, 6 in 801 cases, and the ratio in Polak's series was 1 death in 16 cases. Kornfeld reports a series of 50 cases with no deaths.

The fetal mortality varies from 60 to 95 per cent. Fetal death is chiefly due to asphyxia caused by interference with the uteroplacental circulation.

In my own series there was one maternal death in a patient in whom there was present the Couvelaire syndrome with the hemophilic type of hemorrhage. The history and the course of this case is as follows:

Case 1. Mrs. C. Z., hospital No. 52986, aged twenty-five, para 0, full term. Herp regnancy was uneventful until two weeks before admission to the hospital, when she was struck by an automobile. She fell on her abdomen but complained of no particular pain. There was no vaginal bleeding at the time. The week following this accident there was a slight rise in the systolic blood pressure and a trace of albumin appeared in the urine. On the day of admission, she was suddenly seized with excruciating abdominal pain. The uterus was ligneous, board-like and extremely tender, especially so over its upper right third. She was in profound shock and semiconscious. There was no external hemorrhage. No fetal heart was heard. The fetus appeared big, the presentation was vertex and the head was unengaged. The cervix was thick, one finger dilated and the membranes were intact. Besides abruptio placentae, rupture of the uterus had to be seriously considered in this case. Because of the condition of the cervix, the possibility of rupture of the uterus and the patient's desperate state, immediate delivery was decided upon. A classical cesarean section was performed under gas oxygen ether anesthesia. The uterus was found to be hard and board-like and bluish in hue (not a typical Couvelaire uterus, but of a deeper blue than the normal appearing uterus). On opening

the uterus a large fetus was found and delivered stillborn, the placenta was free, and the cavity of the uterus was filled with clots. While closing the incision in the uterus, a steady ooze of blood of watery consistency was noted. The patient's general condition was such that a Porro operation was considered out of the question. She was given a citrate blood transfusion immediately following the operation, but failed to rally.

I was particularly interested in the question of recurrence of abruptio. The great majority of the articles published on abruptio make no mention of the subject of recurrence. Of the standard obstetrical textbooks, only DeLee touches on the question and that in but one sentence, "Abruption may recur in subsequent labors." Two of the largest maternity hospitals in this country could furnish me with no statistics on the matter of recurrence.

In this series of 15 cases, I have delivered 5 women subsequent to abruptio. One of these patients had a recurrence. Her history is as follows:

Case II. Mrs. E. R., aged twenty-four, Para i. One year ago she had a premature separation of the placenta at full term. She was told by her family doctor at the time that she had kidney trouble. I was called in consultation and with conservative treatment she was delivered by me of a stillborn infant. She made a good recovery. She became pregnant shortly afterwards, was on a diet that contained very little protein and showed no evidence of toxemia such as rise in blood pressure or albumin in the urine until about two weeks before term. Her pressure then rose moderately and small amounts of albumin appeared in the urine. On the day of admission to the hospital she began to bleed and had abdominal pain of moderate severity. Her condition was fair. The fetal heart could not be heard. The uterus was board-like, the cervix two fingers dilated and the membranes intact. I decided to treat her conservatively. I ruptured her membranes, packed her and gave her small doses of pituitrin. The bleeding, however, continued and her condition grew worse. I therefore gave her a transfusion and performed a transperitoneal extraperitoneal cesarean section under local infiltration anesthesia. The uterus presented the typical picture described by Couvelaire as uteroplacental apoplexy. The fetus was dead. The placenta was lying loose and the uterine cavity was filled with blood clots. The uterus contracted in a satisfactory manner and I therefore closed the uterine and abdominal incisions. Her convalescence was marked by the development of a pelvic abscess, which I opened and drained per vaginam. She left the hospital in good condition.

I was also very much interested in the question of pregnancy and parturition following abruptio, a subject concerning which not one of the articles and textbooks I read mentioned even a single word. As already stated I have personally delivered 5 women following abruptio, one of whom had a recurrence. The significant data in regard to the other 4 are as follows:

Case III. Mrs. R. E., para i, aged twenty-seven. Her previous labor four years ago was complicated by abruptio and eclampsia. Her present pregnancy was uneventful except for some staining or very scant bleeding early in her pregnancy. This subsided after rest in bed and calcium therapy. Blood pressure, urine and blood chemistry determinations were normal throughout the pregnancy and the puerperium. She delivered a living baby spontaneously.

Case IV. Mrs. S. H., para i, aged twenty-five. Five years previously, I performed a cesarean section on this patient because of a very grave form of abruptio. The uterus was an extreme example of the Couvelaire type. Her intervening history was irrelevant except for an obstinate peripheral neuritis. Her pregnancy was uneventful, the urine and blood pressure were normal throughout. I delivered her of a living child by classical cesarean section.

Case V. Mrs. R. W., para i, twenty-four years old. I had performed a classical cesarean section under local anesthesia on this patient two years previously, because of abruptio and toxemia of pregnancy, as evidenced by a blood pressure of 190/100, four plus albumin and granular casts in the urine. In addition there was present an atresia of the vagina and cervix, making vaginal delivery impossible.

Two years later, I did a classical cesarean section under general anesthesia. I performed this operation before the onset of labor. On opening the peritoneal cavity, I found a hole in the uterus about the size of a silver half dollar near the fundal end which was plugged by the amniotic sac. The uterine muscle about this area was very thin and stretched. I delivered a normal living infant and sterilized her. She made an uneventful recovery. During this pregnancy her blood pressure and urine were normal.

Case vi. Mrs. C. G., para i, aged twenty-three. During her previous pregnancy at about term she developed a toxemia of pregnancy and a moderately severe form of abruptio. I ruptured her membranes and introduced a bag into the cervix and she delivered spontaneously a stillborn child.

One year later, I delivered her normally of a normal living infant. Her urine and blood pressure were normal throughout pregnancy. She is now pregnant again.

In this series the diagnosis was in doubt in only a very few instances. In the fatal case, I seriously considered rupture of the uterus but even in that case I was more inclined to the diagnosis of abruptio. Placenta previa had to be excluded in some cases and conversely abruptio had to be excluded in some cases of placenta previa. The textbooks also include in the differential diagnosis such conditions as intra-abdominal injuries, gallstone colic, rupture of an appendiceal abscess, perforation of the gall bladder or of a gastric or duodenal ulcer and rupture of an ectopic pregnancy.

The most serious complication in this series was a profuse postpartum hemorrhage. The patient received an immediate direct blood transfusion and made an uneventful recovery. One patient developed a peripheral neuritis which was very annoying and lasted for about two years.

The treatment of the condition was so well summarized by Polak that I will repeat it here. As soon as the diagnosis of abruptio is made, treatment must be instituted. The plan of procedure to be adopted will depend upon certain obstetric factors: i.e. (1) the period of gestation, (2) the parity of the woman, (3) whether or not the woman is in labor, (4) the condition of the membranes, (5) the condition and amount of dilatation of the cervix, (6) the amount of blood loss, (7) the general condition of the patient, and finally, (8) upon the presence or absence of infection. The general indications are to empty the uterus and control the hemorrhage. Polak very strongly emphasized and stressed the point that no operative procedure be undertaken on a bleeding case before a blood transfusion is given. Hofbauer, I believe, is mainly responsible for introducing into obstetrics and popularizing the transfusion of blood prior to operation and the employment of local in preference to general anesthesia in these cases. Many of these cases if not a direct result of toxemia, are certainly associated with toxemia, and general anesthesia in such patients still further injuries the kidney and liver.

I have tried in this series to employ the conservative plan of treatment wherever possible. This consisted in an initial dose of morphine, artificial rupture of the membranes, tight vaginal packing and hypodermic injections of small doses of pituitrin when the labor pains were weak or absent.

My practice now in the grave cases is to transfuse and then perform cesarean section under local infiltration anesthesia.

In this series 5 patients delivered spontaneously. I delivered 2 by low forceps, one by internal podalic version and breech extraction, one by breech extraction; in 2 patients I introduced bags and both delivered spontaneously; I performed cesarean section 5 times. I encountered the Couvelaire type of uterus five times. Polak seemed to favor cesarean section followed by hysterectomy in these patients. The fatal case was the one case in which I considered hysterectomy but her condition in my opinion was too grave to attempt it. I have subsequently delivered 2 of these 5 patients of normal living infants by classical cesarean section.

DISCUSSION AND CONCLUSIONS

This series is entirely too small to warrant the drawing of definite conclusions. This paper was written with the idea in mind of stimulating observation and reports and the building of a statistical structure. The conclusions here drawn are in no wise to be considered final or binding. They just strike me as being reasonable at the present time.

As to the etiology of the condition, I find it hard to see how one can doubt that toxemia plays a major role in the production of abruptio.

In the treatment of the Couvelaire type of uterus, it seems to me that in the majority of cases hysterectomy would be too radical a procedure. I believe that this type of uterus should be watched for a minute or two and if it shows contractile power it should be saved. The uterine muscle is apparently damaged in these cases and in subsequent labors, those women who were treated by cesarean section should again be similarly treated, preferably before the onset of labor.

The patient who died came under my observation before transfusion prior to operation and local anesthesia were recognized procedures in these cases. At the present time, I would transfuse in such a patient prior to operation and perform the operation under local infiltration anesthesia. If the bleeding or oozing could not be controlled, I would not hesitate to amputate the uterus.

Pregnancy was observed in 5 patients in this series after abruptio. Four of the women had an uneventful pregnancy and labor, and gave birth to living children. In one patient abruptio recurred and she was delivered of a stillborn fetus.

A woman who once had abruptio need not necessarily be advised to avoid pregnancy for her chances of having a normal pregnancy and labor are good.

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[For Remainder of References see p. 298.]

ANTIVENIN IN THROMBOCYTOPENIC HEMORRHAGE

IMPORTANCE OF BOTHROPIC TYPE

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THE first report (by Dr. H. C. Clark, Director of the Gorgas Memorial Laboratory, and the writer) of the use of the antivenin of bothrops atrox (*fer de lance*), the South and Central American snake, in the treatment of purpura hemorrhagica[1] has been followed by the publication of three additional case records. Analysis of these reports, with the addition of a second case by the author, comprises the scope of the present paper.

CASE PROTOCOL

Mrs. J. B. S., married, aged thirty-seven, was admitted to the Anglo-American Hospital, Havana, on October 8, 1931. The history was of epistaxis of two days' duration, unrelieved by home measures. Slight but frequent bleeding from the gums over a period of several years was also recorded. The rest of the history and the general physical examination were irrelevant. The condition of her gums appeared normal.

The patient was a well-nourished woman, pallid and anxious. Her general condition was good, temperature, pulse and respiration normal, blood pressure 135/80. There was a constant hemorrhage from the left nostril. Inspection revealed the entire left side of the nasal septum as the site of capillary hemorrhage. Tannic acid 5 per cent and adrenalin were applied to the bleeding area without effect. Anterior and posterior nasal tamponage was accordingly performed.

The blood count revealed erythrocytes 4,250,000; hemoglobin 65 per cent; leucocytes 6600; neutrophiles 72 per cent. Bleeding time (ear) sixteen minutes; clotting time four and one-half minutes. The platelet count was 117,000. Blood chemistry and blood Wassermann tests were normal.

On the following day the nasal packs were removed, with immediate recurrence of hemorrhage. When the packs had been in position, only partial hemostasis had been secured. The left side of the septum was cauterized with crystalline chromic acid, and the packs reapplied. Control of bleeding was definitely secured at this time.

On the third hospital day, little bleeding was evident. The patient feared removal of the packs, however. Bothropic antivenin, 10 c.c., was administered intramuscularly, with a slight reaction.

Fourth day: packs removed and left out; insignificant nasal bleeding; blood platelets 130,000; bleeding time six minutes. Bothropic antivenin 5 c.c. (reaction).

Fifth day: platelet count 180,000. Antivenin 7 c.c. (reaction).

Sixth day: platelet count 172,000. Bothropic antivenin 4 c.c. The patient was discharged from the hospital.

Convalescence was marked by sloughing of the septal mucous membrane. There was no recurrence of bleeding. On the fourteenth day, the platelet count was 230,000; bleeding time six minutes. The patient remained in good health during the next four months, when she was lost to observation.

Summary of Case: Capillary epistaxis of moderate severity; thrombocytopenia; prolonged bleeding time with normal coagulation time; restoration of thrombocytes and bleeding time to normal following injections of bothropic antivenin.

CASE REPORTS IN THE LITERATURE

An analysis of the salient features of all cases known to the writer in which antivenin has been employed is contained in Table 1. The clinical courses of these patients will be briefly presented.

CASE 1. *Purpura hemorrhagica* (Smith[2]). Platelet count 60,000, increased to 153,000 following exsanguination-transfusion. Epistaxis and purpura uninfluenced by transfusion. Platelets fell to 60,000 the following

day. Crotalic antivenin administered. Bleeding t opped in twenty-four hours. Platelet count 366,000 in two days. "Phenomenal action." Recovery.

Case II. *Gingival hemorrhage; thrombocytopenia* (Smith[2]). Platelet count 56,000. Transfusion, 800 c.c., had no effect on gingival hemorrhage, and platelet count declined to 50,000. R.B.C. 1,210,000, hemoglobin 20 per cent, W.B.C. 6600 when crotalic antivenin was injected. No effect was observed on bleeding or platelet count, the latter falling to 33,000 when death ensued on the fifth day.

Case III. *Post-tonsillectomy hemorrhage; thrombocytopenia* (Stockton and Franklin[3]). This patient had had severe nasal bleeding following a submucous resection five weeks prior to tonsillectomy. After the tonsil operation, sublingual and perilaryngeal blood extravasation required the performance of tracheotomy. This incision was reopened for hemostasis. Severe nasal bleeding recurred nineteen days after tonsillectomy, and lasted for four days, uninfluenced by blood transfusion. The patient was in collapse and not expected to live with platelet count 40,000, when two desensitizing doses of bothropic antivenin were given. These caused local hemorrhage and extravasation. Ten cubic centimeters of the antivenin were then injected. The bleeding stopped within twenty-four hours. Twelve days after the administration of antivenin the platelet count was 272,000. Recovery was uneventful.

Case IV. *Purpura hemorrhagica* (Taylor[1]). A fulminating case, with bleeding from all mucous membranes and uncontrollable epistaxis. The patient was in a collapse condition, and preparations were under way for an emergency transfusion when the similarity

Table I

Analysis of all reported cases: antivenin in hemorrhagic states

Reporter	*Sex & Age*	*Diagnosis*	*History*	*Blood Count*	*Platelet Count*	*Bleeding Time*	*Clinical Course*	*Result*
1. Smith	♂ 7	Epistaxis; purpura; thrombocytopenia.	5 days chills, fever, epistaxis.	R.B.C. 2,780,000 Hb. 50% W.B.C. 8800	60,000	.....	No benefit from transfusion. Crotalic antivenin given. Bleeding stopped in 24 hours. Platelet count 366,000 in 2 days.	Recovery
2. Smith	♂ 28	Gingival hemorrhage; thrombocytopenia	Duration undetermined	R.B.C. 1,210,000 Hb. 20% W.B.C. 6600	50,000	.....	No benefit from transfusion. Crotalic antivenin given on 4th hospital day with no effect on platelets or bleeding.	Death
3. Stockton & Franklin	♂ 23	Post-tonsillectomy hemorrhage; thrombocytopenia	Severe nasal hemorrhage 7 wks. before	R.B.C. 2,100,000 Hb. 40% W.B.C. 20,000	40,000	24 hrs.	No benefit from transfusion. Collapse. Bothropic antivenin 10 c.c. Bleeding stopped in 24 hours. Platelet count 272,000 in 12 days.	Recovery
4. Taylor	♂ 21	Purpura hemorrhagica	Fever; epistaxis; hematuria 7 days	R.B.C. 2,800,000 Hb. 45% W.B.C. 7600	.....	9½ min.	Collapse. Bothropic antivenin on 2nd hospital day. Bleeding stopped in 2 hours.	Recovery
5. Taylor	♀ 37	Epistaxis; thrombocytopenia	Epistaxis 2 days	R.B.C. 4,250,000 Hb. 65% W.B.C. 6600	117,000	16 min.	4 injections antivenin. Platelet count 230,000 on 16th day.	Recovery
6. Cubas	♂ 24	Purpura hemorrhagica; leucopenia	.....	.....	.....	.....	Multiple transfusions of no benefit. Collapse. Two injections crotalic antivenin without effect.	Death
7. Bronfin & Singerman	..	Arsphenamine aplastic anemia; hemorrhages	.....	.....	.....	.....	Bleeding time reduced after antivenin.	Death

of his symptoms to those of severe poisoning from the bite of the *fer de lance* suggested the trial of bothropic antivenin. Epistaxis and gingival bleeding ceased within two hours after the injection of 10 c.c., and recovery without relapse ensued.

Case VI. *Purpura hemorrhagica; leucopenia* (Cubas[4]). Cubas administered two injections of crotalic antivenin to a critically ill patient. Severe reactions were experienced after both injections. Repeated blood transfusions failed to modify the pernicious course of this case, which terminated fatally on the thirty-sixth day. (The type and severity of leucopenia are not stated.)

Case VII. *Aplastic anemia* (Bronfin and Singerman[5]). To combat a purpuric tendency in a case of aplastic (arsphenamin) anemia, the authors used a single injection of antivenin (type not specified) with marked reduction in the bleeding time, but without effect upon the course of this inevitably fatal anemia.

PRESENT STATUS OF TREATMENT IN THROMBOCYTOPENIA

Lack of precise information relating to the cause and pathology of thrombocytopenia, or more particularly of purpura hemorrhagica, predicates empiricism in the therapeutic approach. Jones and Tocantins,[6] for example, believe that the clinical manifestations of purpura hemorrhagica may be introduced by platelet reduction, capillary hyperpermeability, or a combination of these factors. High protein diet, liver therapy, ultraviolet radiation and viosterol should receive consideration in treatment after initial blood transfusions. With failure to effect permanent improvement by these agencies, splenectomy or ligation of the splenic artery must be considered. The reliance placed by these writers upon blood transfusion in the acute fulminating type of hemorrhagic purpura is not supported by the experience of Smith[2] and Stockton and Franklin[3] in the cases cited, nor by the controlled clinical observations of McLean, Kreidel and Caffey.[7] In their cases, splenectomy was reserved for the most severe, and yielded the result of common experience in the acute fulminating cases: a mortality of 50 per cent. The general results of cases treated by ultraviolet therapy alone, blood transfusion alone, and transfusion followed by splenectomy are so indistinguishable that the authors are reluctant to draw conclusions, although transfusion and splenectomy are regarded as of some value. Removal of infective foci, when feasible, is advocated by these writers, as well as by Jones and Tocantins. Emphasis is directed toward the fallibility of the platelet count in prognosis, the comparative frequency of spontaneous remissions, and the capricious nature of the disease.

ANTIVENIN THERAPY

In the light of conflicting evidence advanced by the therapeutic measures in larger series of cases, it is presumptious to make claims for antivenin therapy. Careful study of the clinical courses of Cases I, III and IV, however, would indicate more than a casual relationship between the introduction of antivenin and prompt recovery in apparently hopeless cases. Provided sensitization to horse serum is not extreme, a trial of bothropic antivenin should, in the writer's opinion, be made in all cases before splenectomy is undertaken. In fulminating cases, it may be given intravenously, the usual precautions against protein shock being observed, in a dosage of 10 c.c., repeated several times in twenty-four hours if necessary, or at daily intervals.

BOTHROPIC VS. CROTALIC ANTIVENIN

Recent investigations by Steiner and Gunn[8] as well as those of Phillips, Robertson, Corson and Irwin[9] substantiate the normal fluctuations in thrombocyte count, and the surprising degree to which they are influenced by a variety of internal and external stimuli. These studies suggest that factors other than platelet deficiency contribute to the production of purpura hemorrhagica and thrombocytopenic hemorrhages.

It is known that the venom of certain South American snakes (genus bothrops) possesses the faculty of disturbing capillary permeability to a greater extent than does that of the North American serpents (rattle snake, copperhead, mocassin). The bite of the *fer de lance* produces hemorrhagic phenomena almost constantly, whereas Hutchinson,[10] in an analysis of 306 cases of snake bite poisoning on the North American continent, reported hemorrhage in only 62 (20.2 per cent). The predication of antihemorrhagins and antihemolysins in the antiserum of genus bothrops (bothropic antivenin) gives this antivenin preference over crotalic antivenin in their application to thrombocytopenic hemorrhagic states.

CONCLUSIONS

1. An additional case of the successful treatment of thrombocytopenic epistaxis by bothropic antivenin is presented.
2. The usefulness of this agent is suggested by a review of reported cases.
3. The probable superiority of bothropic over crotalic antivenin when used for this purpose is indicated by a consideration of the characteristics of South and North American snake venoms.

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*Continued from p. 271.

TREATMENT OF ACUTE EMPYEMA

AN INSTRUMENT FOR CLOSED DRAINAGE*

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ALTHOUGH the treatment of most diseases, at one time or another in the history of medicine, has ranged anywhere from the utterance of mystic words to the arcane powers of herbs and stones and metals, still empyema seems to have enjoyed a surgical niche from the earliest times known.

The science of medicine seems to be endowed with an eternal pendulum-like swing which gives us the waxing and waning tides of interest now in this and now in that particular subject. It should not be surprising, therefore, that medical men, in the enthusiasm for the new, sometimes forego measures which, although employed without scientific grounds, still, in the light of today's knowledge, seem to conform, with slight modifications, to the perfect desideratum.

The treatment of acute empyema illustrates this point.

Druitt[4] says:

> Paracentesis thoracis . . . is an operation . . . for empyema. An incision . . . is made through skin and muscles, and the point of the bistoury to be passed through the pleura. If fluid escapes from this puncture, a trocar may be plunged in, or if there is no doubt in the diagnosis, a trocar may be employed at once. It may be advantageous to employ the "drainage tube" which M. Chassaignac has adopted for large abscesses in general, and which has been used in empyema by Dr. Goodfellow and Mr. DeMorgan. This is a small India-rubber tube, with many perforations, passed through the cavity of the abscess. . . .

From the last decade of the last century up to a few years ago open drainage for acute empyema reigned supreme. During and following the World War opinions differed. The pendulum has again swung back to the closed method.

Let us scrutinize a few elementary facts and see the wisdom for closed drainage.

As we know, the lung is an elastic structure which is held expanded by the negative intrathoracic pressure. An accumulation of pus in the pleural cavity must of necessity impinge on the size of the lung and, since two objects cannot hold the same place at the same time, the lung, which is of contractile tissue, must necessarily become compressed, and therefore collapsed, in proportion to the amount of fluid present. If air be now allowed to enter the pleural cavity, and open drainage certainly accomplishes this, the negative intrathoracic pressure is eliminated and the lung becomes still further collapsed.

As the mediastinum is a flexible structure the negative intrathoracic pressure of the opposite side pulls it over, thereby compressing somewhat the lung on the sound side also. We see then that open drainage, although it allows for a speedy evacuation of pus, causes collapse of the lung on the involved side, partial collapse of the opposite lung, and shock due to "mediastinal fluttering" during inspiration and expiration, which, because of the diminished aerating area, become more frequent and decrease oxygenation of the tissues with the resultant partial asphyxia of the vital centers. Need we wonder at the high mortality which, according to Lockwood,[11] ranges from 50–70 per cent in children and 20–30 per cent in adults?

Although it was known that the heart could be displaced by the pressure of the fluid, "in which case the gradual evacuation by aspiration is preferable," to quote

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McFadden,[12] still the mobility of the mediastinum did not become apparent until its demonstration by Graham and Bell,[7] which fact established the rationale for the substitution of the closed method for the wide open pyopneumothorax. This, says Hedblom,[9] is "perhaps the most fundamental and important contribution to the treatment of acute empyema since Hippocrates." Why? Because the removal of the fluid by the closed method not only obviates the sequelae of the open pyopneumothorax but considerably decreases the convalescence as it allows for gradual re-expansion of the collapsed lung by virtue of its elasticity and the negative intrathoracic pressure, thereby tending to obliterate completely the empyema cavity.

And yet, as late as 1921, DaCosta[3] even though he states that "in some cases" following rib resection and drainage "the discharge ceases and yet pulmonary function is not completely restored" still on the preceding page he emphatically says: "I do not believe in any of the appliances to drain with a small tube and maintain negative pressure in the chest to favor lung expansion."

However this method has come to stay. Authorities are today unanimous in admitting that many patients are certainly cured by closed drainage alone.

Closed drainage may be accomplished by:

1. Multiple aspirations as practiced in the pre-Listerian era, which can seldom be counted upon to effect a cure *per se*.

2. Multiple aspirations followed by the substitution of an equal amount of air through the same syringe which, recommended for tuberculous empyema, is now employed successfully in the non-tuberculous types by Danna[2] of New Orleans.

3. The introduction of a trocar and catheter. This method, we have seen, was first introduced by Dr. Goodfellow and Mr. De Morgan in 1859.[6] It was later re-discovered and recommended by Thiersh[14] to be subsequently forgotten and lately acknowledged as the procedure of choice.

My discussion and explanation of the thoracotome deals with this last method. Packard[13] emphasizes the necessity of not allowing any air to enter the chest. Now it must be admitted that in introducing a catheter through an ordinary trocar atmospheric air is unavoidably aspirated into the pleural space even through Bettman[1] in a recent article claims that with a little practice "the catheter can be inserted into the trocar in a split second." The same objection can be raised in the employment of his modification of Professor Forschwach's thoracotome (Fig. 1) in which a silver tubing is used around the catheter to prevent the muscles and ribs in older children and adults from obliterating the lumen of the catheter.

By means of my thoracotome not only is the introduction of the catheter made absolutely air-proof, but it allows a metal tubing to be left in place in the same air-proof way.

A glance at the photographs and diagram of the thoracotome will at once show its simplicity. The thoracotome (Fig. 2), which to be exact ought to be named thora-trocar-otome, is fashioned after a simple trocar and comes in two sizes: one for adults which permits the close fitting of a catheter size No. 24 F., and one for children which allows the introduction of a No. 16 F. catheter. The cannula is made of two pieces (A-B) which fit snugly at their proximate ends, and these are held together by a screw (S-S1). The proximal piece of the cannula (B) has a side arm of the same diameter at an acute angle of 15 degrees for the easy introduction of the catheter, and the female part of the screw (S1) which is prevented from slipping off by an enlargement (E) at the male end of that piece of the cannula. The distal piece of the cannula (A) has a rectangular metal plate (P) with the male part of the screw (S) attached to it. At the end of this part of the screw are two needle holes (H) for tying the catheter to it by means of a heavy silk thread. The distance between the end of the cannula and the metal plate in the

adult size is 2 inches; in the one for children it is 1½ inch. The rectangular plate serves the purpose of preventing that piece of

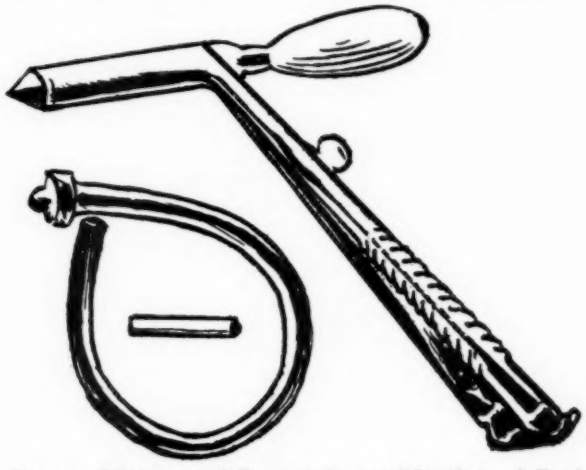

Fig. 1. Diagram of Bettman's modification of Prof. Forschwach's thoracotome with Pezzer catheter and silver tube. (Silver tube is actually larger than catheter, which is not evident in this illustration.)

the cannula from slipping into the empyema cavity and, at the same time, to fasten it to the chest wall with strips of adhesive. The stilet is of solid metal to give consistency to the thoracotome during its insertion. It has, 2 inches from its sharpened end, a groove to mark the length of it to be withdrawn to allow the full introduction of the catheter.

Acute empyema is never so "acute" as to demand emergency drainage. Unless the fluid is frankly purulent no intervention should be contemplated. If its accumulation is so profuse as to cause severe pressure symptoms, aspiration of small amounts of the fluid is consistent with good judgment. Streptococcal empyema is notoriously tardy in becoming purulent, but even when the fluid is purulent from its onset, as in the pneumococcal type, no drainage should be done until the active pneumonia has completely subsided. Therefore, unless the general condition of the patient demands a few aspirations, drainage by the thoracotome method should be delayed until the empyema is well walled off.

Method of Using the Thoracotome

It having been proved by needle aspiration and smear examination of the pus that the empyema is not tuberculous, a site for its drainage is selected. This of course depends on the type of empyema. If the

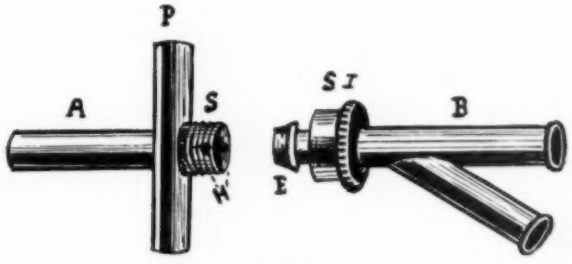

Fig. 2.

empyema is non-interlobar, in which case we can either wait until it spreads to the interparietal pleural space or aspirate it under the fluoroscope, and if it is non-encysted, when the site for drainage is fixed and undisputed, then two places are open for selection: (1) the seventh, eighth or ninth interspace in the posterior axillary line as practiced by the majority, and (2) the sixth interspace in the midaxillary line, as suggested by Hutton and seconded by DaCosta.

The reasons for the second (quoting DaCosta) are:

> (1) It is over the portion of the lung which expands last. An empyema is drained only partly by gravity, and most of the fluid is really forced out and the cavity obliterated by lung expansion. If an incision is made anterior or posterior to this point the expanding lung will block the drainage opening and a pus cavity without drainage will remain in the midaxillary line. (2) Such an incision permits a patient to lie on his back without making pressure on the drainage tube.

Another reason which may be given is that it allows for irrigation without the minimal disturbance of the patient, especially if he is asleep.

The portal of entry having been selected, the skin is cleansed as for a routine operation. A 1 per cent solution of novocaine with a few drops of 1:1000 adrenalin is injected in the skin and then deeply into the muscles up to and including the pleura. This is highly desirable to prevent the rare but none the less embarassing "pleural

reflex." With a bistoury the skin is niched to facilitate the introduction of the thoracotome. The latter is then slowly and

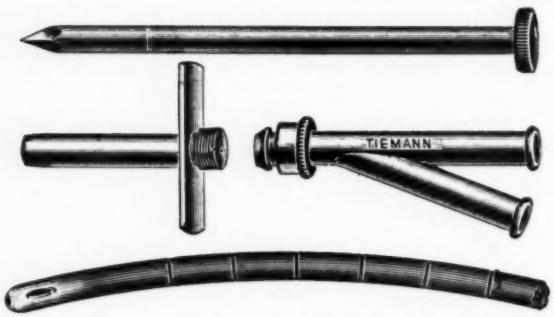

Fig. 3. Author's thoracotome with stilet and modified catheter.

steadily thrust into the empyema cavity, care being taken that in its passage it lies close to the upper border of the lower rib so as to avoid injuring the nerve and blood vessels on the under surface of the rib above. The corresponding catheter, with its distal end clamped, is inserted into the side arm and the stilet withdrawn until the groove in it is reached to permit then the further introduction of the catheter into the empyema cavity. The screw is now loosened and the proximal piece of the cannula withdrawn slightly to allow for the application of another clamp at the exposed part of the catheter. The clamp at the end of the catheter is then removed, the stilet withdrawn and the proximal piece of the cannula slipped over the catheter through its side arm. The catheter has no flaring end; it has a terminal opening and one-side fenestration, and is marked by circular grooves ½ inch apart. The purpose of these grooves is the progressive shortening of the catheter in the empyema cavity as the latter gradually becomes obliterated. A black silk thread inserted through the two needle holes in the male part of the screw is now tied around the chosen groove of the catheter to hold it from slipping inward or outward. A few pieces of split gauze, as the individual case demands and which depends on the robustness of the patient, are then placed between the skin and the metal plate, and the latter held in place by long strips of adhesive. A light binder as described by Bettman is very useful and is recommended.

In infants where the size of the inter-

Fig. 4. Complete set showing adults' and children's sizes with corresponding catheter and special clamp.

costal space does not permit the introduction of the thoracotome, the method as used by Danna ought to prove worthy of execution unless it be desired to resect a small piece of the rib under local anesthesia and then use the thoracotome.

The postoperative care is tedious and requires religious vigilance and scrupulous technique.

Lavaging the empyema cavity has been recommended for the past five decades. A number of solutions were advised among which have stood out hydrogen peroxide in 50 per cent solution, bichloride of mercury solution 1:5000 and carbolic acid 1:100. It was Lewachew,[10] however, who advised the use of the innocuous sodium chloride solution. He said: "After withdrawal of a small portion of the exudate the same quantity of salt solution (0.06–0.07) is introduced into pleural cavity. By repeating this operation several times the entire exudate may be replaced by saline solution." This also embodies the modern

conception of irrigation.

During the World War Dakin's solution was employed. It has the good features of lessening putridity and dissolving the flakes of fibrin in the exudate which have a tendency to obstruct the catheter.

Probably the best course would be to use physiological salt solution until plugs of fibrin tend to obstruct the drainage when the employment of Dakin's solution for a few times will thoroughly liquefy the contents of the empyema cavity.

In children it is better to withdraw a small amount of pus for a few consecutive times before instituting the regimen of irrigation.

This regimen may be carried out by the syringe method as advised by Hedblom and Bettman, or a "Y" glass tubing may be attached to the catheter, one arm of which leads to a graduated bottle containing the irrigating solution suspended 2 feet above the patient, and the other arm to a tube either clamped or ending below the surface of an antiseptic fluid in a jar at the bedside.

This latter method I advise while the patient is bedridden, whereas that of Hedblom and Bettman is especially applicable during convalescence when the patient is up and about.

It has been recommended that the irrigations be done every two or three hours, but this, as well as the amount of the irrigating solution to be left in the empyema cavity, is a personal equation and can, therefore, vary. One thing must be emphasized however, and that is that the irrigating solution should never be allowed to go in with any force as it may break the tender adhesions.

In the presence of a bronchial fistula, which can be demonstrated by a straight fluid line in an *x*-ray plate or by excessive coughing during the irrigating, it is best to dispense with the irrigations for two or three days. It will be found that the fistula usually closes following the simple drainage of pus. The irrigations can then be resumed.

In double empyema it is safer to do a closed drainage on one side and multiple aspirations on the other until such a time as the empyema on the side of closed drainage is well under control. Then closed drainage can be instituted on the opposite side. By this procedure, I believe, many seriously ill patients, and especially children, can be managed to safety who might otherwise succumb to the overwhelming infection.

A word must be said about chronic empyema.

I do not wish to be interpreted as stating that closed drainage is curative in all cases of acute empyema. I have already said that authorities are today unanimous in admitting that *many* patients are cured by closed drainage alone. This means that there is a certain percentage of cases wherein the lung, for obvious reasons, fails to expand completely. A small cavity is therefore left unobliterated to which the word "chronic empyema" is affixed.

For this latter condition the operations of Lilienthal, Schede, Estlander, or Ransohoff, according to the gravity of the condition, must be performed to effect a cure.

CONCLUSIONS

1. The treatment in general of acute empyema is discussed. Realizing and respecting individual peculiarities a few minor points in the management of these cases have been purposefully omitted and left to individual discretion.

2. A simple instrument is presented which permits not only the introduction of a catheter in an air-proof way, but also allows at the same time the utilization of a metal tubing in the intercostal space to prevent the ribs and muscles from constricting the lumen of the catheter.

3. A modified catheter is also described.

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[For Remainder of Bibliography see p. 298.]

REPEAT OF A RARE CONGENITAL MALFORMATION OF INTESTINE

WITH CASE REPORT

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IN June, 1931, I reported[1] an uncommon congenital atresia of the rectum, entire colon and about 12 inches of the ileum, occurring in a baby girl born to apparently normal white parents in Honolulu, T. H.

For comparison the Pathological Report is repeated.

Abdomen: Autopsy record follows: On opening abdomen no free fluid or adhesions found. All organs are in normal position.

Gastrointestinal Tract: "The stomach and duodenum are about normal size. The upper jejunum is somewhat distended and bluish red in color. The ileum and entire colon are pale in color and small. The ascending, transverse and descending colon measure 16 in. in length. The sigmoid colon and descending colon are small, being between 5 and 6 mm. in diameter. On opening, canal is patent, will admit a very small probe, and contains a small amount of grayish, sticky mucoid-like material. The cecum is slightly larger than the descending colon, contains more material similar to sigmoid and measures 9 mm. in diameter. The ileum is about 8 mm. in diameter. Thirty-two inches above ileocecal valve the intestines are somewhat constricted, being about the size of sigmoid, then there are 6 in. that approach normal size; and again there is a constriction. The upper jejunum measures 3 cm. in diameter and contains normal appearing, thin, yellowish feces. Other organs not removed. Those in abdomen appear normal.

Anatomical Findings: 1. Malformation, congenital, colon and ileum (narrowing). 2. Intestinal obstruction, due to 1.

Microscopical Sections: Microscopical sections from sigmoid colon, ascending colon and ileum show mucosa present and apparently normal. Two muscle coats present, which also appear normal, but on stretching to the size of normal gut the muscle and mucosa are extremely thin, and evidently lack power. H. R. Livesay, Major, Medical Corps.

FIG. 1. Congenital atresia of colon with peritonitis. A, Stomach, turned up. B, Upper duodenum. C, Lower duodenum, cotton in lumen. D, Ileum with dry muconium. E, Cecum, with appendix. F, *Atresic colon.* G, Rectum at site of obstruction. H, Anus, plugged with cotton. I, Gall bladder. K, Urinary bladder.

After the first defective child the mother asked me if I thought she could have a normal baby. I told her that the condition of the first baby was rare, and that a second child would undoubtedly be normal.

About fourteen months later she gave birth to another child with practically the same malformation.

Through the courtesy of Dr. E. A. Fennel of the Clinic, Honolulu, I am permitted to publish their record and autopsy findings in the second child in order that

[1] Metcalfe, R. F. An uncommon congenital malformation. AM. J. SURG., n.s. 12: 532, 1931.

the report of the two malformed babies, born to the same parents, may be made a matter of record.

Fig. 2. Cross section of atresic colon, showing serosa, longitudinal and circular muscle fibers, submucosa, muscularis mucosa, tunica propria, mucosa and patent, though collapsed, lumen.

A white woman of twenty-seven years went through a normal pregnancy with no unusual symptoms save marked distention from excessive amniotic fluid. This somewhat underdeveloped and undernourished, II para, went into labor at term, and at the end of three hours was delivered of a male child. The head and shoulders were delivered with ease, but difficulty was experienced with the abdomen, which was much enlarged. The child cried normally and seemed in all other respects, with the exception of the protruding, tense abdomen, quite normal. The anus was normal, but the examining finger in the rectum met a total obstruction about 2 inches above the anus. X-ray confirmed this fact.

Eight hours after birth the infant was given a spinal anesthesia of spinocaine, ⅛ the adult dose, with apparently perfect anesthesia. A median incision permitted the removal of more than a pint of deep yellow fluid containing many fibrinous floccules, and brought to view the fact that the entire abdominal contents were firmly cemented together by a strong fibrinous deep yellow exudate, that covered

all the organs, and suggested in appearance well-scrambled eggs.

The rectum was reduced in size and as the examination was continued upward the sigmoid was found to have the diameter of a cigarette and about the same consistency to the palpating finger. An incision into it was made, a catheter inserted into the rudimentary lumen, pushed upwards for about 3½ inches, and the abdomen was closed. Five hours later the child seemed to be in great pain; seven hours postoperative it died.

Cultures of the abdominal contents, made at time of operation, proved sterile.

At autopsy the contents of the thorax and abdomen were removed, in toto, fixed and later dissected. Figure 1 illustrates, better than words, the findings. No abnormalities were found in the esophagus, lungs or heart. There was no exudate in the pleural sacs. All surfaces of the liver were covered by the yellow fibrinous exudate; this exudate on the under surface on the right, closely binding together the liver, kidney and cecum (which was pulled up high), was more dense and organized, suggesting older material. The pylorus felt hard, but on section seemed normal. The lower portion of the duodenum was dilated, thin-walled and empty. The jejunum and ileum were markedly distended and filled with meconium, soft in the upper portions, quite hard and putty-like as one approached the cecum. The walls of the small intestine were thin; they seemed simply distended rather than hypertrophic. The cecum was large and the appendix rather prominent.

The large intestine, from its very beginning, showed a marked atresia, throughout it had about the diameter and consistency of a cigarette. In the upper portions the walls were quite thin, the lumen, 0.75 cm. filled with meconium that was brown rather than black. This lumen, after the first 7 cm. dwindled into a thread-like aperture that could be followed by the brown stain which marked its course. This lumen was lost in the upper rectum where it could no longer be traced. The anus was normal, as were the gall bladder, ducts, kidneys, ureters and bladder.

Histologic sections of the small intestine, just above the cecum, showed normal structures save that the epithelium was badly eroded. Histologic sections of the colon in several areas, as the accompanying photomicrograph (Fig. 2) illustrates, showed relatively normal structures. The mucosa and tunica propria were normal. The submucosa contained frequent eosinophiles, the circular muscle fibers might be interpreted as hypertrophic, though that point would be controversial. The serosa was normal. There were no evidences of an inflammatory process in the intestine nor in its mesentery; lymphocytes and leucocytes were conspicuous by their absence.

The evident maturity of the exudate in the area of the caecum and ascending colon made us suspect a perforation at that point; careful search however, failed to reveal any such gross lesion.

There are various theories regarding the etiology of the defective development of the aforementioned children, yet none of them will satisfy the disappointed mother.

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NEWBORN BABY WITH MASSIVE UMBILICAL HERNIA

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NEW ROCHELLE, N. Y.

UMBILICAL hernia in the newborn is unusual. Massive umbilical hernia in the newborn is very unusual.

FIG. 1.

FIG. 2.

Following is the report of a massive umbilical hernia in a newborn baby. It was successfully repaired.

I saw a female baby in consultation twelve hours after it was born. The mother was a primipara; delivery was normal; birth weight was 6 pounds, 13 ounces.

A hernia, 7 × 6 × 10 cm., protruded through the umbilicus. The tied end of the umbilical cord was attached to the hernia. The hernial sac consisted of thin, transparent, glistening tissue continuous with the peritoneum. Coils of small intestine could be seen within the sac.

The baby had been given small quantities of water which she had retained. She seemed to be comfortable.

Attempts to reduce the hernia being unsuccessful, I advised immediate operation.

Under ether anesthesia, given by the drop method, the hernial sac was opened and the neck of the hernia found to be only 0.75 cm. in diameter and the segments of small intestine tightly adherent to the neck throughout its entire circumference. Now it was clear why the hernia had not been reducible before operation.

A circular incision of the skin and anterior rectus sheath around the umbilicus was made, the rectus muscles retracted laterally and the peritoneum opened. Finding it impossible to dissect the intestines entirely free from the neck of the sac I carried the incision around it and split it transversely. The intestines, with the neck of the sac still attached, were then placed in the abdominal cavity. The rest of the sac was tied off and removed. There had been approximately 3 feet of small intestine within the hernial sac.

The hernia was repaired by overlapping the fascia according to the Mayo method and the wound closed without drainage.

The baby withstood the operation well; there was very little interference with respiration.

After the operation she was given 100 c.c. of normal saline solution subcutaneously twice daily for three days, and two intramuscular injections of whole blood, 40 c.c. at a time. She was also given water by mouth on the day following operation, ¼ ounce every two hours.

She vomited only the first few feedings of water. Twelve hours after operation she passed some mucus from the rectum; thereafter she passed normal stools.

The temperature rose to 102.8°F. by rectum on the second day after operation and stayed up through the third day; it dropped to normal on the fourth day.

For the first forty-eight hours after operation the abdomen was moderately distended but was soft at all times. With the establishment of normal stools the abdominal distention grad-

ually disappeared and the abdomen looked much like that of a normal baby.

For the first few days after operation the baby gained strength and had a normal cry.

On the third day after operation the skin on the tip of the nose became red and began to peel. Within forty-eight hours the skin of the entire body became similarly affected. A diagnosis of pemphigus neonatorum was made. The baby lost strength, vomited frequently, required gastric lavage several times a day and had frequent soft, frothy bowel movements. She died on the fifth day after operation.

A complete autopsy was not allowed but the abdominal cavity was reopened. Following is the report of Dr. McIlroy, pathologist:

General Appearance: The body is that of a well-nourished, white, newborn female infant. The epidermis has partially sloughed. The slough involves the face, neck, buttocks, groins and the upper two-thirds of the thighs. It extends over the perineum and vulva. Purulent discharge is present between the labial folds. A transverse incision is present in the region of the umbilicus, securely held together by silk sutures.

Abdomen: When the abdomen was opened no free pus was found. The intestines were smooth and glistening. A bland inflammatory process was found in the region of an inverted portion of the umbilicus.

Anatomical Findings: (1) Superficial sloughing of the epidermis (pemphigus). (2) Operation (umbilical hernia).

Had it not been for a severe pemphigus neonatorum, it seems probable that the baby would have recovered from the herniotomy. The abdominal wound was healing kindly at the time of death and there was no infection within the peritoneal cavity.

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The American Journal of Surgery

PUBLISHED MONTHLY BY PAUL B. HOEBER, INC., 76 FIFTH AVENUE, NEW YORK, N. Y.
FOR THE AMERICAN JOURNAL OF SURGERY, INC.
Editor: THURSTON SCOTT WELTON, M.D., F.A.C.S., NEW YORK

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EDITORIAL

EDITORIAL MEMORANDA

AUGUST is the time for the mountains and the sea shore; a time for fishing, tennis, boating, hiking, travelling, and a period when the duffer, with grim determination, tries to break 100 on the golf course. Therefore, to ease the sweltering weather from our mind, it will not be amiss to touch lightly on many subjects. From time to time we jot down a memo for an editorial. To do such an editorial calls for special reading, delving into old books and dry statistical reports . . . all of which we fully intend to do but never seem to get around to actually doing. Anyway, it's too hot and humid for sustained effort and the following enlarged memoranda may, at some future time, stimulate you to do justice to the subjects and as a result prompt you to submit to us the editorial we seek.

COST OF MEDICAL CARE. This was jotted down several months ago. But why continue the subject? Organized bodies, editors of medical and surgical journals scathingly criticised the "report of the majority," and, with the return of prosperity we venture the prediction it will continue to remain dead in the official archives. But touching on prosperity . . . we feel it is here. Don't you have that feeling, too? We are one of those who did not vote for President Roosevelt but we are convinced he has done a grand and glorious job so far, and if he can continue the pace he has set for himself 60 per cent, we'll vote for him the next time, if we both are alive.

HOSPITAL COSTS. This is an amateur opinion. So, if you happen to be an expert on these matters do not deluge us with

reams of figures. We feel it costs too much for the average person to receive hospital care. We do not refer to the ward patient; neither does the well-to-do patient who can afford a private room, day and night nurses, etc., concern us. We are concerned with young people, married, just keeping their chin above the financial torrent, people of small incomes, but who are used to the usual nice things of life; these people often are cultured and gentle folk; every physician has this problem. A member of this family gets ill and is ordered to a hospital. At best they can afford a bed in a two or four bed semi-private ward room. When we hear (and know for a fact) that such accommodation costs from 6 to 8 or 9 dollars a day (and to this add laboratory, *x*-ray, anesthetic, operating-room, and special drug fees) we get an idea of the money burden this class of citizen must assume. Everything else has gone down but, for the most part, hospital costs have declined very little. Is it because during the Inflation or Boom Era hospitals built a superstructure which added to the overhead and now cannot get rid of that superstructure? Is it because the usual directors, who so gladly made up the annual deficit, are now bent and broke, and red ink gives the hospital authorities nightmares . . . therefore, one must get all and more than the traffic can bear? Maybe this does not apply to small rural hospitals, we can not answer for them, but it does hold true for many of the hospitals in the large cities. Lord, how we'd welcome someone solving this problem for us, even though they convinced us that hospital costs for the middle classes are truly quite low.

Medical Journals and Books. It would be foolish to deny that subscribers of medical and surgical journals and buyers of medical books have fallen off during the recent Period of Gloom. We can truthfully say (and we get this from the Circulation Department) that this Journal has enjoyed but a slight decrease in circulation. We know the Publishers have been concerned, in the main, with the reasons why the circulation has not continued as it used to . . . that is, increase "by leaps and bounds." It has been amazing the way medical publishers have carried their burdens. The last month we hear things started on the up grade. Business is picking up. The sun has come forth from behind the black clouds. People are being recalled to their former jobs. And physicians are resubscribing to journals they cancelled under the fear of continued perilous times. Which is good news. Without the leading medical publishing houses what a sorry state the profession would find itself in. These leading publishers, not only the house of Paul B. Hoeber, Inc. but all reputable publishing houses, merit support. This support will be gained by doctors of medicine buying needed medical books and subscribing to medical and surgical journals, and not reading them only because they are in the "library."

This day we talked with an editor of a semi-medical publication. Naturally, we asked him for constructive criticism of this Journal. His answer included nearly all high class journals. He said, "You all need personality . . . something different." "What, for instance?" we asked. "Oh, pictures, news events, cartoons, jokes, . . . all of you are too scientific . . . doctors are interested in other things besides scientific problems."

We doubt if he is right. You do not subscribe to this Journal to be amused. We try to interest you in things surgical. We know no one would dream of reading an issue from cover to cover. But if we can in each issue publish something that will interest each reader, then we think we've given value received.

The late Mr. Curtis, owner of *The Saturday Evening Post*, is reported to have said: "Should we have an issue of the Post and a reader found everything in that issue interesting to him . . . then, I'd consider it a poor number." True words. But if you have constructive criticism do let us have it. We are woefully hungry for ideas.

T. S. W.

OLIVER WENDELL HOLMES

[1809-1894]

AMERICAN PHYSICIANS

OLIVER WENDELL HOLMES

OLIVER WENDELL HOLMES was born at Cambridge, Mass. on August 29, 1809. His family was one of the "Brahmin caste of New England."

He prepared at Phillips (Andover) Academy. He was of the "famous class of '29" of Harvard. He wrote the class lyrics for the anniversary dinners for thirty-eight years, closing with the touching "After the Curfew."

Holmes then turned to medicine. He went to Paris (1833) and devoted himself to his studies. He returned to Boston in 1835 and went into practice with but scant success. However, he won prizes for medical papers he wrote, and lectured on anatomy at Dartmouth.

In 1847 he was appointed professor of anatomy and physiology in the medical school at Harvard. Holmes called it "not a chair, but a settee in the school." He delivered lectures until 1882. The writer's father was one of his pupils ('68) and confirmed the often printed statement to the effect that Holmes lectured at the end of the day because he was so witty and interesting he held the attention of the students who otherwise might nod or fall asleep.

In 1836 he was the Phi Beta Kappa poet at Harvard. He christened the publication *The Atlantic Monthly* and, as Howell afterwards said, he "not only named but made" it. Each issue of the first volume had a paper of the Autocrat of the Breakfast Table. These popular papers carried the magazine through the financial panic of 1857. In 1858 these 12 papers appeared in a volume. The Autocrat was succeeded by the Professor at the Breakfast Table (1859). In 1871 the Poet appeared. Holmes' novels were "Elsie Venner," "The Guardian Angel," and "A Mortal Antipathy."

He wrote two "witty" papers on "Homeopathy and its Kindred Delusions" (1842); also, a paper on malarial fevers of New England. In 1843 appeared his epoch-making essay on the Contagiousness of Puerperal Fever. This brought him personal abuse, but in time he was honored as the discoverer of a great obstetrical truth.

In 1840 he married Amelia Lee Jackson.

He received honorary degrees from Cambridge, Edinburgh, Oxford and Harvard.

Oliver Wendell Holmes died October 7, 1894 and was buried in the cemetery of Mount Auburn.

T. S. W.

[From Fernelius' Universa Medicina, Geneva, 1679.]

BOOKSHELF BROWSING

SIR ROBERT JONES AND MR. OWEN THOMAS

GEORGE W. HAWLEY, M.D.

COLORADO SPRINGS, COLO.

AMONG the effects of the late Virgil P. Gibney of New York was a small photograph of Mr. Hugh Owen Thomas and his nephew, Sir Robert Jones. Through the courtesy of the family of Doctor Gibney, the writer was privileged to have an enlarged copy made of the original photograph.

This is a striking picture of two striking figures, two figures preeminent in a field of surgery to which they so greatly contributed, two figures idolized by orthopedic surgeons in every land the world over. The names of Owen Thomas and Robert Jones are permanently engraved in the history of surgery.

The pose is characteristic of the men. It is characteristic of master and pupil in the older sense of the master, the teacher and loyal disciple, the succeeding master surgeon. Thomas, life friend of the Liverpool dockmen, is wearing his Liverpool dockman's cap and with him is his ever-present companion, his cigarette. Jones in his easy, quiet manner is attentively listening.

Little did Thomas dream what a part the simple classic splint he designed for immobilization of the knee in tuberculosis was to play in a World War, and what an important part his nephew was to play in that war and in the adaptation of his splint to the treatment of war fractures in the armies of all nations. No one can compute what this one thing accomplished during those war years. No one can estimate the number of Thomas splints forged and used. No one can measure the amount of suffering it spared countless men, the number of limbs and lives it saved, its value to those who were called upon to treat the wounded.

During the closing days of the war, at lunch in a Paris restaurant, one of this country's distinguished surgeons related the following anecdote: He said that it had been his privilege to see what had since remained in his mind as the most conspicuous example of skillful surgery it had been his good fortune to witness. The Society of Clinical Surgeons was on a visit to Edinburgh. This small group stopped en route in Liverpool. It was there he saw Robert Jones. He saw Jones operate one morning long on a long series of varied and difficult cases with a skill, a facility and a technical finish such as he had never seen. This is the Robert Jones, who, in characteristic fashion, with characteristic modesty, when describing removal of the

semilunar cartilage of the knee, remarked that he felt qualified to speak, because he had performed this operation somewhat more than 5000 times.

Fig. 1. Hugh Owen Thomas and Sir Robert Jones.

Hugh Owen Thomas was born in 1834 and died in 1891. His father was a noted Welsh bone setter living in Liverpool. He studied in Edinburgh, London and Paris. In 1857 he was made a member of the Royal College of Surgeons.

Robert Jones was born in Rhyl, June 26, 1858, and was graduated from the Liverpool School of Medicine. He became associated in practice with Thomas at 11 Nelson Street in Liverpool. The house, 11 Nelson Street, will long be memorable to those who have journeyed there. In 1916 the British War Office established the Department of Military Orthopedics under Lieutenant-Colonel Robert Jones. His rapid promotion to the rank of Major General followed. He was knighted before the end of the war and created a baronet a few years later by King George v.

The death of Sir Robert Jones occurred on January 14, since this article was written. He died suddenly of a heart attack at Llanfechain, Montgomeryshire, England. Sir Robert had served as president of the British Orthopedic Association and the British Association of Surgeons. Many universities and many foreign countries had conferred honors upon him. From this country he received the Distinguished Service Medal in 1919 while on a visit as a guest of the American College of Surgeons.

BOOK REVIEWS

OBSTETRICS AND GYNECOLOGY. Edited by Arthur Hale Curtis, M.D. Vol. 1. Phila., W. B. Saunders Co., 1933.

The first volume (1165 pages) of a three volume work on obstetrics and gynecology, edited by Dr. Curtis, is at hand. The leading specialists in these and allied fields have been pressed into service and, if the initial volume is an adequate specimen upon which to form final judgment, then they have produced an excellent work.

It is not our task to consider whether the profession or the times warrant such an ambitious and pretentious undertaking. Also, we have a complex against works by many authors, edited by someone, presented in several volumes, because, in the last analysis, they usually are hodge-podge in contents and style. From a survey of the present volume these faults are not present. Whether or not the authors (or, all medical authors) write in the same style, or Dr. Curtis has accomplished the impossible in editing, in that he has used his blue-pencil lavishly and whipped the whole into a uniform mass, one cannot tell from the outside. However, unless one knew the various chapters were written by different individuals, one could easily arrive at the conclusion it was the work of one man. We could be hypercritical and point out certain subjects that might well be omitted, a chapter here and there that might be left to the formal textbooks (chapters that must be studied by undergraduate medical students but which are skipped by the man in practice) but to accentuate microscopic blemishes would not be fair, for the canvas as a whole is very worth while. The general practitioner (especially the general man who has to do obstetrics) will find it valuable; the trained gynecologist and obstetrician will discover much between the covers to his advantage and, on the whole, find it satisfying reading.

In Volume 1 Franklin H. Martin begins the work with a short chapter on The New Surgical Literature. Then follows Irving S. Cutter's Historical Account of these specialties. This covers two hundred odd pages. Dr. Cutter has a pleasing style; he makes history live; he intrigues the reader with the easy and friendly manner with which he treats of what many imagine is a dry subject. We feel it is a shame one has to invest in this voluminous and expensive work just to find joy in this historical account. Would that the publishers might some day see their way clear to reproduce it as a separate monograph.

Beginning the more formal part of the book we have chapters on the Anatomy of the Female Genital Tract, The Physyology of the Reproductive Organs (Exclusive of Pregnancy); The Morphology and Physiology of Pregnancy, Physiology of the Birth Processes, Labor, Pathology of Pregnancy, and the volume ends with an ample Index.

We would like to list the various authors and tell of their work in detail but the limitation of space forbids. We must center our congratulations on Dr. Curtis who has whipped the various ingredients into a finished product of a high order of merit.

THE PRINCIPLES AND PRACTICE OF OTOLOGY. By F. W. Watkyn-Thomas and A. Lowndes Yates. Balt., William Wood & Co., 1933.

A concise and well-written textbook of otology written with the needs of the practitioner rather than the student in mind. Extensive reference is made to the literature and full credit is given therefor. This book will supplement rather than replace the many more complete presentations of the subject already on the market.

HOLT'S DISEASES OF INFANCY AND CHILDHOOD, A Textbook for the Use of Students and Practitioners. By the Late L. Emmett Holt, M.D., and John Howland, M.D. Revised by L. Emmett Holt, Jr., M.D. and Rustin McIntosh, M.D. Ed. 10, N. Y., D. Appleton and Co., 1933.

The tenth edition of the famous Holt's Diseases of Infancy and Childhood is revised by L. Emmett Holt, Jr., the son of the original author, and Rustin McIntosh, Carpentier Professor of Diseases of Children at Columbia. The work has been brought thoroughly up-to-date. The sections on nutrition are practically rewritten. Holt's is probably one of the few

books that may rank with Osler as the undoubted standard in its field and this edition will maintain the reputation established throughout the previous nine editions. First published in 1897, the book in its ten editions will represent a history of the advances in pediatrics during the past thirty-six years. In the hands of its present editors, a continuation of its old-time popularity for many years to come may be looked for.

Precis D'Anatomie Pathologique. By Gustave Roussy, Roger Leroux and Charles Oberling. Paris, Masson et Cie, 1933, 2 vols.

These two volumes present a concise, authoritative and up-to-date textbook of pathological anatomy. It is well written, splendidly illustrated and remarkably low in price. In contradistinction to many foreign books, this one is fairly well indexed. The lack of bibliography or references for further reading is distinctly noticeable.

Vorbeugung und Bekampfung der Operationsgefahren. By Prof. Dr. M. Kappis. Leipzig, George Thieme, 1933.

This book treats operative surgery from an unconventional angle. The question of the ability of the patient to withstand operation and the measures to be taken to ward off the pitfalls and dangers of operations are here discussed both thoroughly and authoritatively on the basis of years of experience and special studies along these lines.

This work may be read and reread to great advantage by all active surgeons.

Arteriosclerosis, A Survey of the Problem. Publication of The Josiah Macy, Jr. Foundation, Ed. by E. V. Cowdry. N. Y., Macmillan Co., 1933.

This work by 23 authors edited by E. V. Cowdry is, as the title indicates, a "survey" of the problem of arteriosclerosis. The present status of each phase of the subject is outlined by the authors who are allowed to express themselves broadly and freely. This, therefore, furnishes a splendid source book for anyone attacking this problem. The status of the researches in arteriosclerosis to the end of 1932 is summed up in this volume. The suggestion is made that other problems be treated in the same manner as an encouragement to research workers.

Much illuminating material is contained in this volume. It will be interesting to note what use will be made of this book and whether it will further the study of this perplexing problem.

Die Bruchoperation nach Bassini. By Prof. Attillo Catterina. Illus. by Dr. Maler Horatius Gaigher. Berlin, Urban & Schwarzenberg, 1933.

Dr. Catterina, for many years an assistant of Dr. Bassini, has undertaken in this work to present the present status of the Bassini operation. Feeling the need of outstanding illustrations, he was fortunate in having as an associate Dr. Maler Horatius Gaigher, who is a splendid artist and who has produced 16 illustrations in color showing in explicit detail the various stages of the operation.

Prof. Eiselsberg of Vienna in his Preface tells of over a million cases that have already been cured by the Bassini operation. This would furnish in itself a justification for the publication of this volume which will be found extremely practical and convenient for continued reference by those doing this operation.

Surgical Anatomy. By C. Latimer Callander, M.D. With a Foreword by Dean Lewis, M.D., Phila., W. B. Saunders Co., 1933.

A new and up-to-date "Surgical Anatomy" with 1280 illustrations is not a work to which justice can be done in the compass of a short review. Suffice it to say that this book promises to become a classic. Strictly up-to-date with a modern viewpoint, the work shows evidence of the most painstaking care in its preparation. For both student and experienced surgeon, this book will be an indispensable work of reference.

Surgery of the Stomach and Duodenum. By J. Shelton Horsley, M.D., F.A.C.S., LL.D. St. Louis, C. V. Mosby Co., 1933.

A concise and well-illustrated discussion of the subject based largely on the author's own experience. There are 14 chapters and 136 illustrations. While greater elaboration of detail

would be desirable in many of the chapters, the book will be found a handy work of reference.

THE OPERATIVE STORY OF CLEFT PALATE. By George Morris Dorrance, M.D. Assisted by Enayat Shirazy, D.D.S. Phila., W. B. Saunders Co., 1933.

Beginning with a 9 page historiography of the cleft palate and ending with an 87 page bibliography, this work indeed tells "the operative story of cleft palate," and does it well.

There are 534 well-selected illustrations. Anatomy, operative technique and instruments are illustrated wherever necessary but there is no padding.

This book is a complete and authoritative reference work on an important subject.

HIRNCHIRURGIE ERFAHRUNGEN UND RESULTATE. By Dr. Hans Hoff and Dr. Leopold Schonbauer. Foreword by Dr. Otto Potzl. Leipzig, Franz Deuticke, 1933.

In almost 500 pages with 238 illustrations, the authors give their experiences in brain surgery during the last four years at the Vienna Clinic. The work is a distinct contribution to the subject and will be read with interest by everyone doing brain surgery.

THE CONTROL OF FOOTBALL INJURIES. By Marvin A. Stevens, M.D., and Winthrop M. Phelps, M.D. N. Y., A. S. Barnes and Co., 1933.

This book will be of extreme interest to those in charge of football teams but would seem to be of little value to the general surgeon who may see an occasional case of this type and would hardly get much help from the book at that time. The book has a special value in showing the methods of prevention of accidents which, after all, is more than sufficient reason for its publication. One of the interesting points that the authors mention is that the accident rooms in the Yale Bowl "are frequented much more by spectators than players." Every football coach and every doctor in any way connected with a football team should have this book.

COLLECTED PAPERS OF THE MAYO CLINIC AND THE MAYO FOUNDATION. Ed. by Mrs. Maud H. Mellish-Wilson and Richard M. Hewitt, M.D. Vol. 24, 1932. Phila., W. B. Saunders Co., 1933.

In this volume are reprinted 99 of the 484 papers written by the members of the Mayo Clinic staff during 1932, 36 additional papers are given in abstract, 22 are abridged and the titles only are given of the other 327 papers. This volume is quite on a par with the quality, quantity and scope of the previous volumes published annually by this remarkable institution.

ACUTE INTESTINAL OBSTRUCTION

MONROE A. McIVER, M.D., F.A.C.S.

EIGHTH INSTALLMENT

PART II: DIAGNOSIS AND TREATMENT

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Chapter XX

TREATMENT

The general principles governing the treatment of a case of acute mechanical obstruction of the intestine are simple: they consist of relief of the obstruction, and correction as far as possible of the systemic effects of the obstruction. The need for prompt operation has long been stressed; but realization of the importance of the treatment of the secondary effects of obstruction is a relatively recent advance.[1–2] It is now generally recognized that many of the signs and symptoms shown by the patient are due not solely to the absorption of a toxin, as formerly thought, but also to loss of water and sodium and chloride ions from the body by vomiting. The restoration of these substances is an extremely important factor in the treatment,[3] although the amount of replacement needed is, of course, in proportion to the amount of fluid lost, which varies greatly in different types of obstruction.[4–5] *

The management and treatment may be chronologically considered under three heads: treatment before operation; operation; and after care.

Preoperative Treatment

Since promptness in operating is of paramount importance, no preparatory procedure that materially delays surgical relief should be considered. Usually, however, after the decision to operate has been reached there is an interval, while the necessary arrangements for operating are being made, which can be utilized to good advantage in preparing the patient. Proper treatment at this time will often put the patient into much better condition for operation.

Relief of Pain. If the pain is severe, morphine should be administered. Since the diagnosis has been made, morphine

* See May installment, p. 486.

is no longer contraindicated for fear of masking symptoms, and even partial relief from the agonizing types of pain will often materially improve the patient's general condition and relieve the mental anxiety that is often an important feature.

MAINTENANCE OF BODY TEMPERATURE. It is important that any patient prior to operation be adequately protected against exposure. It is especially important in the type of case which shows signs of collapse or is in danger of developing such a condition after operation that the normal body temperature be maintained as a prophylactic against shock.[6] The physician and nurse should be careful to avoid undue exposure during examinations, or while the skin preparation for operation is being carried out, during the administration of enemas, etc. If indicated, the patient may be wrapped in warm blankets and heat applied to the extremities.

TREATMENT OF DEHYDRATION. One of the most striking clinical features of many cases of obstruction is a marked dehydration. This is proportionate to the amount of vomiting that has taken place; for in the vomitus is lost not only water but also the electrolytes sodium and chloride, which are essential in maintaining the normal volume of body fluids. These substances are best returned to the body by the administration of normal salt solution;* as has already been shown in the chapter on body fluids,† no other type of solution will take the place of one containing sodium and chloride.[11] As demonstrated experimentally by Haden and Orr[7] and White and Bridge,[12] water alone, or glucose alone, is without benefit. Not infrequently, however, it may be desirable to add glucose to the salt solution, for the patient may have been starving for some time and the glycogen reserve of the liver be exhausted. Glucose

* In their early work, Haden and Orr advocated the administration of hypertonic saline[7] on the theory that the blood chlorides were low, due to the fact that they were called upon to neutralize a toxin.[8] These authors have abandoned this theory,[9] and it seems physiologically more correct to use an isotonic solution, which also permits the administration of large volumes of fluid. The work of Hughson[10] on the markedly stimulating effect of hypertonic salt solution on intestinal peristalsis is of interest; it would seem unwise, however, to increase peristaltic activity before relief of the obstruction.

† This appeared in the May installment, p. 486.

is usually added in the concentration of 5 or 10 per cent; it is better to use the former concentration if the solution is to be given subcutaneously, for it should be borne in mind that the fluid is not taken up so rapidly from the tissues when it contains glucose, especially if the solution be hypertonic.

Method of Administering the Fluids. Administration of fluids by mouth is contraindicated: they bring on vomiting, which increases the loss of chlorides, and produce distention of the intestinal tract above the point of obstruction (Foster[13] and Gatch et al.). There are a number of methods by which fluids can be given satisfactorily, and these are discussed in the order of ease of administration: (1) by rectum; (2) by hypodermoclysis; (3) intravenously. Where the dehydration is severe, all three methods of administering fluids may be used.

The large amount of fluid that will be absorbed from the rectum when the body is depleted is well known. There is usually no contraindication to administration by rectum, either in the form of a continuous Murphy drip, or by injections of 120 to 180 c.c. (4 to 6 oz.) repeated every three or four hours. Normal salt solution should be used.

The subcutaneous administration of salt solution is one of the simplest and most effective methods for supplying large volumes of fluid to a patient with intestinal obstruction. It may be used alone or in conjunction with the rectal and intravenous methods. It is customary to inject the fluids under the breast or into the subcutaneous tissues of the thigh. It is usually possible in the adult to give 1000 to 2000 c.c. in the course of a single injection without producing too great distention of the tissues: the flow of fluid must be clamped off from time to time when the rate of entrance markedly exceeds the rate of absorption.

Injection of normal salt solution (with or without the addition of 5 to 10 per cent glucose) directly into the venous circulation, is the most rapid and often the most satisfactory method for administering fluids. The usual and well-known precautions for intravenous injection of any fluid should be observed; the

solution should, of course, be sterile, chemically pure, and free from any particulate matter; and it is desirable that it be made up only a short time before its injection. The fluid should be injected at body temperature; and care should be taken that it be not given too rapidly. The amount to be injected depends upon the extent of the dehydration, the size of the individual and his toleration of the treatment. If the fluid is to be given during a short period of time (within half an hour, for example) it is perhaps best to limit the amount to from 500 to 1000 c.c. If a longer time is available, larger volumes can be given.

Gastric Lavage. Patients with obstruction who are having the regurgitant type of vomiting, or where there are other reasons for suspecting a dilated stomach, should have a tube passed and the stomach emptied. Often this not only temporarily improves the general condition of the patient, but renders vomiting during the operation less likely. This latter point is of particular importance if a general anesthetic is to be used, for the patient may regurgitate and aspirate the septic material into the lungs, thus setting up a diffuse, fulminating pneumonia; or he may even aspirate so much material that he dies on the table from asphyxia. The danger from this serious complication is not completely removed by emptying the stomach before operation, since the stomach may rapidly fill again from a dilated intestine. In the more serious cases a small tube may be passed into the stomach through the nose and be left there during the operation.

Anesthetic. The anesthetic to be used must be fitted to the individual case. In Table xiii are shown the types of anesthesia used in cases of intestinal obstruction (exclusive of strangulated external hernia and neoplasms) at the Massachusetts General Hospital during the twenty-year period 1908–1927 inclusive, and the attendant mortality. The low mortality shown by the cases receiving ether is in agreement with the figures recently reported by Miller.[14] As regards the Massachusetts General Hospital figures, the high mortality

attending local anesthesia as contrasted with general, is probably explained on the basis of the fact that the cases received early for operation were generally done under ether; whereas the more desperate types of cases, usually those received late, were usually operated upon under novocaine anesthesia.

Ether. There are many early cases where ether is a safe anesthetic, and the complete muscular relaxation that it produces is desirable from the point of view of a thorough exploration. In the later stages of the obstruction, when the patient is beginning to show systemic manifestations of the disease, ether has a number of drawbacks.

In the first place, shock and collapse are much more frequent after the use of ether, even in patients who seemed to be in reasonably good condition at the time the operation was started. Its action in these cases resembles its effect on patients with severe traumatic injuries, where its use often tends to induce shock if this is not already present, or to depress further the patient who already shows signs of shock. An interesting study of the effects of ether in shock was made by Cattell.[15] This investigator found that a condition of sensitiveness to ether was brought about by any circumstances which tended to depress the general condition of the animals. Upon etherizing the experimental animals there occurred a preliminary drop in blood pressure, probably due to the depressing action of ether on the heart. In normal animals, this was quickly compensated for by the vasoconstrictor action of ether on the peripheral vessels; but when the animals were in shock, no such compensation occurred and the blood pressure continued to fall, reaching zero before the eye reflexes disappeared.

Secondly, the local effects of ether on the bowel may be injurious. Ether normally inhibits the peristaltic activity and decreases the tone of the intestine; and the bowel which is distended and fatigued from obstruction may not resume its activity after operation, even though the obstruction has been relieved. In the less extreme cases, the convalescence may

be rendered more serious by the depressing effect of ether on intestinal activity and tone.

Thirdly, the postoperative pulmonary complications after ether anesthesia should be remembered. Although statistically they are not more common following ether than after other forms of anesthesia, it should be recalled that the more serious cases, which might be expected to have a higher incidence of pulmonary complications, are usually done under local anesthesia. For a recent analysis of postoperative pulmonary complications, see King's article.[16]

Novocaine. Local anesthesia is often the anesthetic of choice in obstruction, particularly in the more serious cases where no extensive exploration is contemplated. There is no depressing effect, either general or local, on any of the bodily functions; and since the reflexes about the throat are intact, the danger of aspirating vomitus is avoided. It has the disadvantage that complete muscular relaxation is not obtained, so that exploration may be difficult; also intestinal manipulation requiring traction upon the mesentery may be painful. It is a form of anesthetic, however, that has a wide field of usefulness in obstruction. Most of the operative procedures required can be carried out satisfactorily without employing supplementary anesthetics. It is particularly indicated in the very sick individual requiring only an enterostomy; and practically all cases of strangulated external hernia can be operated upon satisfactorily without resort to other anesthetics. Its use is by no means limited, however, to those two types of case.

Nitrous Oxide. Nitrous oxide with oxygen is occasionally a satisfactory anesthetic. The gas has little or no depressing action, and actually raises the blood pressure. Unless skilfully given, relaxation of the abdominal muscles is poor, and the straining is likely to push distended coils of intestine out of the wound, and render exploration difficult and dangerous; at times, however, it may be useful as an adjunct to local anesthesia. Because of the danger of aspirating vomitus, the mask should not be strapped on.

Ethylene. Ethylene has proved a satisfactory anesthetic in many cases of obstruction. In some clinics, however, it is not used because of the explosion hazard. Its advantage lies in the fact that it gives better relaxation than nitrous oxide, without having the depressing effect of ether. This anesthetic also may be used advantageously to supplement local anesthesia.

TABLE XIII

TYPES OF ANESTHESIA*

Type of Anesthesia	1908–1917			1918–1927		
	Number of Cases	*Number of Deaths*	*Mortality (Per Cent)*	*Number of Cases*	*Number of Deaths*	*Mortality (Per Cent)*
Ether	99	41	41	99	35	35
Spinal anesthesia	7	1	14	1	0	0
Local (novocaine)	4	2	..	29	20	69
Spinal and general	2	1	..	0	0	0
Novocaine and general	4	4	..	10	6	60
Gas-oxygen	2	0	..	8	6	75
Ethylene	0	0	..	9†	1	11
Total	118	..	..	156		

* Richardson;[17] McIver.[18]

† The use of ethylene has been abandoned at this clinic because of the explosion hazard.

Spinal Anesthesia. Spinal anesthesia is increasing in popularity in many types of gastrointestinal surgery.[19] It is at times a satisfactory and desirable anesthesia in obstruction: it gives good muscular relaxation and has the advantage that under its influence intestinal peristalsis and tone are stimulated. It has, however, certain disadvantages. It interrupts the vasoconstrictor impulses passing by way of the splanchnic nerves

to the blood vessels of the abdominal viscera, often producing considerable fall in blood pressure. In the deeply toxic patient this drop in blood pressure may be profound and serious, for considerable strain on the splanchnic circulation may already exist and anything that tends to abolish the compensatory mechanism may be disastrous. If, therefore, the patient shows any sign of shock or is in such a precarious condition that the onset of shock seems likely, spinal anesthesia should be given only after careful consideration of its dangers.

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Chapter XXI

TREATMENT (Continued)

The Operation

General Considerations. The location and character of the obstruction (particularly as to whether the large or small intestine is involved and whether or not strangulation is present) having first been determined as exactly as possible, it is important to form as accurate an opinion as possible in regard to the patient's general condition. It should be realized that a patient with intestinal obstruction is often a much poorer risk than might be supposed from the objective findings: the compensation that these patients have been able to establish is easily broken by anesthesia and operative procedure, and once this is broken they quickly pass into a state of collapse and shock from which recovery may be difficult or impossible. With this in mind, the type and extent of operation must be fitted to the individual patient. The chance of recovery for the extremely ill patient is better if the minimum amount of operative procedure be carried out; and it is easy to be led into doing too much. Excessive manipulation of the abdominal viscera, especially traction on the mesentery, or evisceration, is to be avoided. Speed is desirable; but precision and gentleness are essential.

In performing the operation the greatest care should be used in attempting to free adhesions or to deliver an adherent loop: a friable, inflamed, distended loop of bowel may be almost as easily torn or punctured as wet paper; and if this accident occurs the result is usually fatal. Even if no gross perforation occurs, many surgeons believe that the permeability of an obstructed bowel is at times so increased that a peritonitis may result from undue handling.

Ordinarily, as soon as the peritoneum is opened dilated coils of intestine present themselves in the wound. Great care is

necessary that they do not escape, especially if the patient makes a sudden straining movement, for not only is such an accident conducive to shock, but the dilated coils may be extremely difficult to return to the abdomen. Care should be taken to cover immediately with warm, moist towels or large gauze sponges any coils of intestine that are exposed.

Choice of Procedure. The first important question to decide is whether the abdomen is to be explored, or whether some simple procedure such as an enterostomy shall be carried out without exploration. The answer to this question may be clear from the preoperative study of the patient; or it may depend upon the operative findings, as for example evidence upon opening the peritoneal cavity of strangulation, which had not been suspected beforehand.

In the seriously sick patients, if there is no evidence of strangulation and the obstruction is in the small intestine, an enterostomy alone is usually sufficient without any exploration; the intestine should not be allowed to come out of the abdomen. If it has been determined before operation that the obstruction is in the large intestine, a cecostomy should be done with a large tube (unless the obstruction is in the ascending colon or at the ileocecal valve, which can easily be determined); no exploration should be done, and the intestine should not be allowed to come out of the abdomen. In doing either an enterostomy or a cecostomy, if the intestine is much distended an aspirating needle attached to a suction apparatus should be inserted to empty the loop that is to be opened.

Aside from the foregoing group of cases, there is the larger group where an exploration is indicated. The hand should be inserted into the abdominal cavity and the experienced surgeon should be able to feel any band, growth or volvulus, in fact anything where there is local distention and thickening of the intestine. In certain instances a collapsed intestinal coil may be followed up to the point of blockage, or a coil of dilated intestine followed down to the obstruction, and the nature of the pathology determined. The situation may be very simple,

as in the case of a single band compressing a loop of gut, or of an annular carcinoma of the colon. On the other hand, it may be extremely puzzling, as for example in cases where the intestine is snarled and rotated around some point fixed by a mass of adhesions. Tracing a section of collapsed intestine may lead one to an opening of an abdominal fossa where an internal strangulation has occurred, or to some hole in the mesentery or omentum through which herniation and strangulation has occurred; palpation in the region of the terminal ileum may reveal an impacted gallstone or an inflammatory reaction around a Meckel's diverticulum which may be responsible for the obstruction; or the characteristic mass of an intussusception may be felt. The intestine should not be allowed to come out of the abdominal cavity during the exploration.

If by this conservative method of exploration it is impossible to locate the obstruction, and if it is imperative that it be found, it may be necessary to resort to partial or complete evisceration. Cheever[1] suggests that the intestines, as they are brought out of the peritoneal cavity, be placed in a large sheet of rubber dam and covered with pads wet with warm saline solution. D. F. Jones[2] says that only in cases of absolute necessity should the intestines ever be brought out of the abdominal cavity, for he believes that even with the utmost precautions this procedure is dangerous to the life of the patient.

Location of the Incision. As regards the incision in a case of intestinal obstruction, in general it may be said that when no definite indication exists as to where this should be made, it had best be located over the lower abdomen or near the midline.[3] A right para-umbilical incision which can be extended upward or downward is often useful and furnishes access to a large area of the abdomen.[4] The great majority of obstructions involve the lower bowel and are to be found in the lower abdomen or pelvis, and the incision should furnish ready approach to this region. The incision should be of sufficient length to permit exploration to be carried on with ease and rapidity.

Character of the Peritoneal Fluid. As soon as the peritoneum is opened, most valuable information may be obtained from the character of the peritoneal fluid. In cases of obstruction there is an increased amount of clear, straw-colored fluid; if the fluid is of a dark, bloody color, a strangulation must be suspected and sought for. In the recent series of 335 cases at the Massachusetts General Hospital there were 21 instances in which blood-stained fluid was present and a gross interference with the mesenteric blood supply found; in a number of instances the fluid was described as foul-smelling, indicating extreme damage to the bowel. There were 4 cases in which blood-stained fluid was present but no definite interference with the mesenteric circulation found; the fluid in these cases was probably the result of the great distention and congestion of the obstructed intestine. There were also a few cases in which, although there was interference with the mesenteric circulation, it was definitely stated that there was no bloody fluid. Its presence must depend upon the degree and duration of the strangulation. It represents an important operative finding; but its absence, particularly in cases brought early to operation, does not preclude the possibility of strangulation.

At times the character of the peritoneal contents may suggest that a mistake in diagnosis has been made: the purulent fluid may suggest some inflammatory disease, or the escape of gas and fluid indicate the perforation of an ulcer.

Orientation. The immediate problem is to obtain some idea as to the level of the obstruction. This may be easy or may be difficult. If a collapsed small intestine can be located, it is obvious that the obstruction is in the small intestine and that it must be sought for at a level higher than the collapsed portion. On the other hand, if, in addition to dilated coils of small intestine, distended colon (easily identified by the characteristic bands and appendices epiploicae or the attachment of the omentum) is found, the obstruction is presumably situated in the colon distal to the distended portion. It is sometimes time-

saving to examine the cecum and terminal ileum first. These structures serve to orient one for further exploration, while their distention or collapse at once furnishes evidence as to

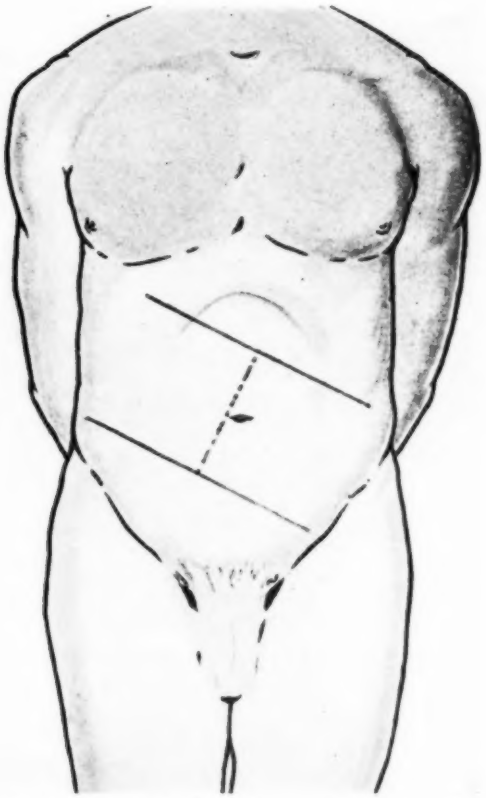
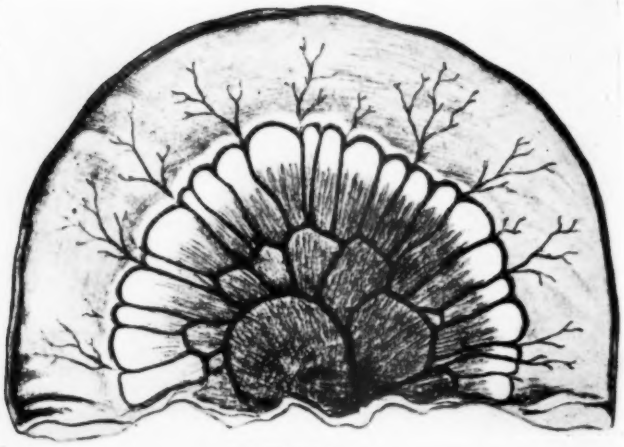


FIG. 52. Broken line is approximately line of mesenteric root as located on abdominal wall. Oblique solid lines divide abdomen into three compartments containing upper, middle and lower thirds of small intestines. (Monks.[5])

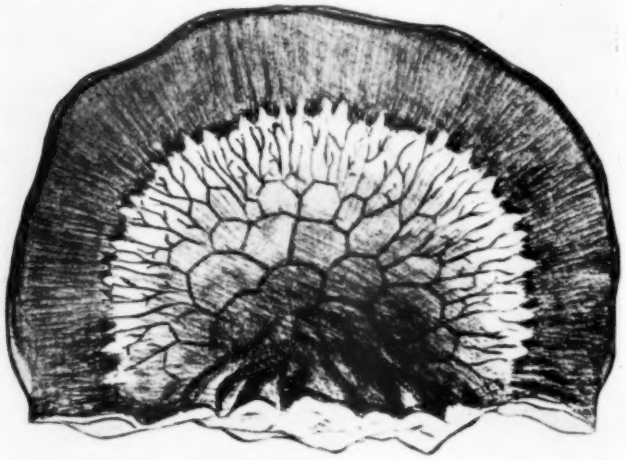
whether the obstruction is located in the large or small intestine.

Monks, in 1903[5] on the basis of the attachment of the mesentery and certain distinctive anatomical features of the intestine and its blood supply, called attention to a number of points that are useful in determining approximately to which portion of small intestine any given loop belongs. Diagrams from his article are reproduced here. In Figure 52 the broken line is approximately the line of the mesenteric root as located on the abdominal wall. The oblique solid lines drawn at right

53A



53B



53C

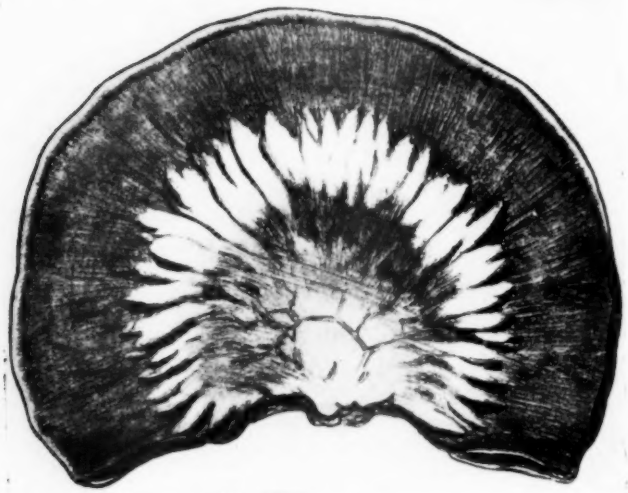


FIG. 53 A, B and C.

angles to this line divide the abdomen into three compartments containing the upper, middle and lower thirds of the small intestine. The correspondence between the different parts of the small intestine and the oblique attachment of the mesentery is obvious. The upper 6 feet or so of the tube are generally confined to the left hypochondriac region. The upper small intestine is in general of larger diameter than the lower portion of the intestine; the walls are thicker, and due to greater vascularity the color is pinker. These distinctive features, however, may be largely obliterated or obscured by the effects of distention. The size and arrangement of the mesenteric blood vessels offer characteristic points and may furnish a clue as to the identity of an intestinal loop; Figure 53 A, B, C illustrates these points. The mesenteric vessels opposite the upper part of the bowel are larger than at any other point, and diminish in size until the lower third of the gut is reached. In general, the mesentery of the upper intestine is thinner and more translucent than that of the lower intestine.

The foregoing points in regard to the approximate identification of any given portion of the small intestine may or may not be helpful in a particular case. They may, however, furnish valuable hints, and the student would do well to note at the autopsy table, in the dissecting room, or during the course of general abdominal work, the small anatomical variations that distinguish different levels; for at times the information may be valuable and prevent unnecessary manipulation in an emergency.

Distention of the coils is a factor which often renders exploration difficult in cases of obstruction. In some of the

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FIG. 53. A. Loop of intestine at 6 feet, showing vasa recta. Secondary loops are a prominent feature. (Monks.[5]) B. Loop of intestine at 17 feet. Mesentery is opaque, and small tabs of fat begin to appear along mesenteric border of gut. Vessels are represented by a somewhat complicated network and are seen with difficulty in thick fat of mesentery. C. Loop of intestine at 20 feet. Gut appears to be thick and large. Mesentery is quite fat and opaque, and large and numerous fat tabs are present. Vessels, which are complicated, are seen with difficulty and are represented by mere grooves in fat.

more extreme cases one will have to decide whether or not to abandon the idea of exploration and simply do an enterostomy; if there is reason to suspect strangulation the exploration must, naturally, be continued. In the course of exploration in very distended cases it may be desirable to empty certain of the more dilated coils. This may be necessary in order to reach the desired portion of the abdomen, to make it possible to bring out a portion of the bowel for purposes of an enterostomy or cecostomy, or to return to the abdominal cavity coils that have been drawn out onto the surface of the abdomen. Owing to the highly septic nature of the intestinal contents in obstruction, however, this is always a dangerous procedure and must not be undertaken lightly. The procedure is best carried out with a needle or small trocar attached by rubber tubing to a suction apparatus; if the aspirating needle is put into the intestine and allowed to drain without suction, leakage around the needle is likely to occur. The puncture wound may be closed with a purse-string suture; gauze packing should be carefully placed to absorb any unavoidable leakage.

Codman,[6] Cheever[1] and others advise in selected cases (in general, late cases of small intestinal obstruction) the insertion into the intestine above the point of obstruction of a "Monks tube" or a stiff, woven catheter, over which the bowel may be "threaded" and the distended intestine emptied of its contents. This procedure is carried out, not only with the idea of reducing the distention, but also in order to draw off the toxic contents of the obstructed intestine as a therapeutic measure. The method, however, is not used in most clinics, and should certainly not be attempted by anyone who has not had wide experience in abdominal surgery.

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Chapter XXII

TREATMENT (Continued)

The Operation (Continued)

Procedures for Relieving the Obstruction. Depending upon the pathology found at exploration, there is great latitude as to the choice of operative procedures. In certain cases, the cause of the obstruction may be directly removed: the division of a constricting band, the untwisting of a volvulus, the reduction of a strangulated hernia or intussusception, may be all that is required and may be very easily carried out. When a direct attack is inadvisable or impossible, the blockage may be indirectly relieved by establishing drainage above the obstruction through an enterostomy or, occasionally, by anastomosis of the intestinal loop above the obstruction to one below, thus side-tracking the intestinal stream around a benign or malignant stricture. Where the intestine has suffered considerably from the effects of obstruction and is dilated and atonic above the blockage, an enterostomy may be indicated in addition to direct removal of the cause of the obstruction, in order temporarily to divert the intestinal stream and prevent overloading and further distention of a portion of bowel that is functionally incapacitated. In cases where extensive interference with the circulation of the bowel exists, resection of the gangrenous bowel and reestablishment of the intestinal continuity may be required in addition to relief of the obstruction.

The operations in the recent Massachusetts General Hospital series were grouped somewhat arbitrarily under the following heads: relief of the obstruction only; relief of the obstruction and drainage; drainage only; resection with immediate or delayed anastomosis; miscellaneous procedures which could not be grouped under the foregoing headings. The results are shown in Table xiv.

It will be noted that those cases in which relief of the obstruction alone was carried out had by far the lowest mortality. These cases were, in general, those that came to operation early in the disease.

Resection. Question as to the viability of a segment of intestine comes up frequently in operations for acute obstruction. The cyanotic, engorged appearance of a strangulated loop of gut is characteristic. It may be easy or difficult to decide whether it is viable and should be returned to the abdominal cavity or whether it should be resected. A wrong decision usually means a fatality. There are a number of points that help one to decide whether the intestine has been damaged beyond the point where it may safely be conserved.

The appearance of the peritoneal coat should be observed: if this has lost its normal sheen and glisten and is of a dull, grayish color with fibrin on the surface, the outlook is not good. Palpation is helpful, for the viable intestine has a certain tone which can be felt and which is replaced in the later stages by a relaxed, sodden feeling. Any evidence of peristalsis should be carefully looked for: if it occurs, either spontaneously or after pinching, it is of course a favorable sign and indicates that the neuromuscular apparatus has not undergone serious degeneration.* The mesentery of the involved segment should be palpated for pulsations in the mesenteric arteries, including the vasa recta at the mesenteric border.[1] The veins should be examined for the presence of clot. If the circulation is obviously inadequate,† the bowel should be resected; or, in the desperate

* Eisberg,[1] however, reports a case in which although peristalsis was noted at operation, the gut later became gangrenous.

† Considerable experimental work has been carried out on animals to determine how much interference with the mesenteric circulation could be produced without resultant gangrene of the bowel. There have also been numerous experiments to determine the point at which ligations of the mesenteric vessels could best be carried out without fatally interrupting the circulation of the bowel.

The early anatomical and pathological studies of Mall,[2] Welch,[3] and Dwight[4] laid the foundation for subsequent work on this subject, particularly as regards the working out of the anastomotic connections in the mesentery and bowel. Most of the more recent experiments have consisted of stripping the mesentery from varying lengths of intestine, or of ligating various mesenteric vessels, and observing the survival period of the animal

cases, the gangrenous intestine may be drawn out onto the abdominal wall and a catheter inserted in the proximal (oral) portion as advocated by Eisberg.[1] In doubtful cases the loop should be wrapped in a sponge with warm saline, and a little time allowed for the circulation to be re-established. Improvement in color and other convincing signs of returning circulation may appear promptly; if they do not appear after a reasonable time, resection should be carried out. There may be an occasional borderline case where one is loath to return to the abdominal cavity a loop of questionable viability, but equally reluctant to carry out the radical procedure of resection of gut that might survive. In such a case, the loop in question may be left outside the abdominal cavity and the peritoneum carefully closed around it. Under these conditions the loop could be promptly removed if it did not survive, and the

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and the pathological reactions of the involved segment of bowel. Eisberg[1] points out that the ligation of the vessels of the second mesenteric arcades (see Monks' illustration, p. 322), has proved safest in experimental work. This author also feels that trauma to the vessels and prolonged spastic muscular contracture of the gut wall is important in preventing the return of adequate collateral circulation. Another factor pointed out as important is the speed with which vascular occlusion takes place, the gut suffering severely when it occurs rapidly, even though a short segment is involved, while interference with the blood supply to considerable areas is much better tolerated if the vascular occlusion takes place slowly.

Bost[5] and Rothschild,[6] repeating older experiments by various investigators,[7] separated the mesentery from varying lengths of intestine and found that the animal survived (in one instance, where 8 inches of the intestine had been separated from its mesentery;[5] the mesentery was sutured back in place and the omentum wrapped about its attachment to the bowel.) It is an old clinical practice to wrap the omentum around intestines whose viability is questionable. The experiments of Scudder[8] and Wilkie[9] dealt with this point. Wilkie says: "From these experiments I concluded that the wrapping of a portion of the intestine, the viability of which is doubtful, in the great omentum is of some value in preventing gangrene and perforation; but it is a practice whose range of usefulness is distinctly limited." Scudder came to essentially the same conclusion. Apparently in some cases vascular connections are made between the omentum and the damaged gut; but in most cases probably the chief value of the omentum is in preventing the spread of peritonitis.

In regard to the clinical application of this experimental work it should be remembered that most of the ligations of blood vessels and separations of the mesentery were carried out on healthy intestines, while in acute strangulation in humans not only is the mesenteric circulation impaired, but often, also, the capillary circulation in the bowel wall is handicapped by congestion and distention due to the intestinal obstruction.

amount of toxic absorption in the meantime would be small;* if on the other hand it proved viable, it could at a suitable time be returned to the abdominal cavity.

Where a small, localized area of necrotic bowel is found, such, for example, as might result from the direct pressure of a band upon the bowel wall, the damaged area may be infolded without resorting to resection.[12,13] Where it is not possible to infold the necrotic area, at times a local excision may be carried out without cutting across the mesenteric border of the intestine; the wound is closed as in an end-to-end anastomosis.

In cases requiring resection, great precaution should be taken (by walling off the operative field with gauze and by careful application of clamps) to avoid spilling any of the intestinal contents and so setting up a rapidly fatal peritonitis. A suction apparatus to remove any fluid may be used to advantage.

After a gangrenous segment has been removed and all bleeding controlled, the question as to the re-establishment of the continuity of the intestine arises. In a number of cases it may be safe, even highly desirable, to proceed at once with an appropriate anastomosis (either end-to-end or side-to-side) by means of suture, or, where time is especially important, by using a Murphy or a Jaboulay button. High fistulae are often poorly tolerated, especially by young children; and at times an immediate anastomosis, although a risky procedure, is better than a delay and secondary operation. In certain cases, however, particularly in dealing with a very sick individual or when technical difficulties to anastomosis exist (as for example when there is great disproportion between the dilated gut above and the contracted portion below), it is wiser not to attempt to carry out an immediate anastomosis but to bring the severed ends outside the peritoneal cavity, closing the peritoneum around them, and wait until the patient has recovered sufficiently before restoring the intestinal continuity. It may be

* The experiments by Travers[10] and White and McIver,[11] show that toxic absorption under these conditions is small even when gangrene is present. (See also Eisberg's case.[1])

useful to tie into the ends flanged glass tubes ("Mixter tubes"; "Paul tubes") to lead off the drainage. In certain cases where drainage is profuse, it is possible later to connect the ends of

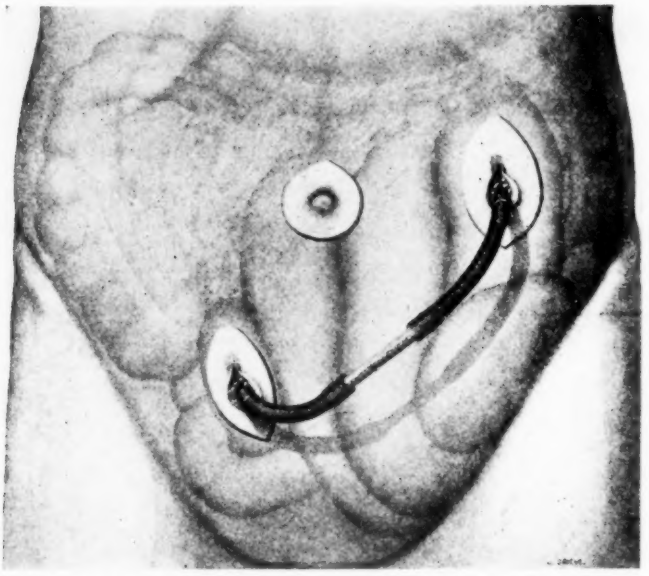

FIG. 54. Device joining enterostomy tubes, permitting secretions from intestine above an obstruction to pass into collapsed gut below. (Wilkie.[14])

the tubes so that the intestinal stream from above passes into the lower bowel for absorption, and dehydration and malnutrition are diminished (see Fig. 54).[14] Or, one may collect the drainage and inject it into the lower segment. These procedures are particularly applicable to a high fistula; if the fistula is low, so that there is a large absorbing surface above, they are usually not necessary. The secondary operation may be postponed for days or weeks, depending upon the condition of the patient or upon how satisfactorily the fistula can be managed. Shedden[15] has recently called attention to the fact that an extraperitoneal Mikulicz type of operation, where the dividing walls between the loops of gut are gradually cut through with an appropriate clamp, may be utilized in establishing the continuity of the small as well as of the large intestine.

In the group of resections in Table VIII, the mortality was high. Once the bowel has become gangrenous there is only a short time interval in which surgery can save the patient; if many hours elapse before operation, so much toxic absorption takes place that removal of the damaged gut is without avail. There were 22 resections in the recent Massachusetts General Hospital series, with 16 deaths, a mortality of 73 per cent. In 9 of these patients the anastomosis re-establishing the continuity of the intestinal tract was carried out immediately; there were 7 deaths. In 13 instances the ends of the intestine brought out and anastomosis reserved for future operation; there were 9 deaths. Enterostomy was combined with resection in 4 instances.

"*Sidetracking*" *Operations.* Indirect operations for relieving the obstruction have already been mentioned. Before taking up the principal one of these, enterostomy, the question of "sidetracking" operations will be considered.

Short-circuiting the intestinal stream around a point of obstruction is particularly indicated where the obstruction is the result of some inoperable neoplasm. Certain cases where the obstruction is caused by such a mass of adhesions and inflammatory reactions that it is unwise to attempt to deal with it directly,[16,17] and others where the obstruction comes about from some benign stricture, may also be advantageously treated by this method.[18]

At times the sidetracking operation is useful as a first step in a two-stage operation, the attack upon the primary cause of obstruction being reserved for a secondary operation after all obstructive symptoms have subsided.

Turner[19] draws attention to the dangers of certain types of exclusion operations, where by lateral anastomosis the ileum is joined to the transverse or left side of the colon because of a malignant growth in the proximal portion of the colon. The author makes the point that a cecostomy should be carried out in this case; for occasionally the cecum, lacking a vent (the ileocecal valve at times preventing any discharge back into

the ileum), will be so greatly distended that it becomes gangrenous or bursts. (See also Morison's article.[20]) Stretton[21] reports a case where, following the transplantation of ileum into the sigmoid, a blind pouch composed of cecum and a portion of colon became so distended that resection was required. Codman's case[22] where a permanent cecostomy was left is of interest.

TABLE XIV

OPERATIVE PROCEDURES

(156 CASES, MASSACHUSETTS GENERAL HOSPITAL TEN-YEAR SERIES)*

Type of Operation	*Number of Cases*	*Number of Deaths*	*Mortality (Per Cent)*
Relief of obstruction, only	68	13	19
Relief of obstruction and drainage of the bowel	27	15	55
Drainage of the bowel, only	33	19	58
Resection — Immediate anastomosis	9	7	73
Resection — Delayed anastomosis	13	9	
Miscellaneous†	6	5	83
Total	156	68	44

* It will be noted that those cases in which relief of the obstruction alone was carried out had by far the lowest mortality: these cases were for the most part those that came to operation early in the disease. The higher mortality in the other groups indicates in general the seriousness of the condition found at operation and is not due to poor choice of operative procedure. (McIver.)

† There were 2 cases in which because of the condition of the patient the operation was abandoned; and one case in which due to a diagnostic error the obstruction was not relieved.

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